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A BIOLOGICAL IONIZATION CHAMBER[1]

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IN the last ten years there have been fifty-nine articles in *Strahlentherapie* and the *Fortschritte* concerning the measurement of the dosage of X-ray. The number hints admirably at the difficulties of the problem. The use of Sabouraud pastilles and of the Kienböck photographic method have been abandoned in deep therapy, though still employed as giving approximate dosages suitable for dermatologic therapy but not accurate enough for the determination of the heavy dosage of highly filtered radiation. The selenium cell has many defects and has to be carefully calibrated. Many varieties of ionization chambers have been proposed, none of which is wholly satisfactory for practical purposes. There remains only the Duane type of open ionization chamber, using a delicate galvanometer as a measuring instrument, and the elaboration of this type of chamber now undergoing extensive investigation at the Reichsanstalt. The Duane instrument is simple, but the galvanometer is an expensive instrument which cannot be handled by anyone without very considerable manipulative skill. The Reichsanstalt instrument is too cumbersome and delicate for practical daily use, and expensive. It is probable that the two machines measure a minute fraction of the energy of the beam of X-ray which passes through them. If the effects of radiation are due solely to the absorbed energy, as every physicist must believe, and if the biological object is of dimension sufficiently small so that it has an absorption comparable to that of the column of air in the ionization chamber, it is probable that the lethal effects of such radiation should be parallel with the absorption measurements. In my laboratory it has been shown that, using two varieties of biological material—animal tumors and *Drosophila* eggs—the effects are parallel with the ionization produced in such an open ionization chamber. Experiments in my own and other laboratories, though not definitely conclusive, point to the fact that these chambers measure energy, and that that energy, so measured, is independent of the wave length. We can set aside the question of the different sizes of quanta carried by the different wave lengths of radiation and such degradation effects as those discovered by Compton, for the long waves produced according to Compton's notions must have the same ionizing effects on the column of air as they do in the minute biologic ionization chamber—the egg of *Drosophila* (Fig. 1). The reactions are measured not by the galvanometer but by the life or death of the organism. What the absolute sensitivity of this egg is, is unimportant, except that as a matter of experimental convenience it is well

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to have something which is highly sensitive rather than highly resistant.

It is for this reason we have employed the eggs of *Drosophila*. As a practical ionization unit they cost only an infinitesimal amount as compared with any commercial ionization apparatus; they do not have to be calibrated, and the eggs from wild as well as those from homozygous strains show year after year exactly the same death-point to equal quantities of radiation. The technic of growing the flies is simple. A few can be captured in warm weather by exposing a piece of banana at a window, or samples may be obtained from any zoological laboratory. If a sufficient supply of eggs to permit of tests being made every day are obtained, some six dozen pint milk bottles are sufficient for the necessary flies. The bottles are washed with hot water and plugged with a gauze-covered mass of cotton. Over the bottom of the bottle is poured half an inch of fly food made up as follows: Fifteen grams of powdered agar is dissolved by heat in 500 c.c. of water; 120 c.c. of molasses and 105 c.c. of Karo syrup are added, and the whole boiled; 240 grams of yellow corn meal are then stirred in, and water added up to 1,500 c.c. This quantity is sufficient for 15 bottles.

After the mixture has solidified, four or five drops of a thick watery suspension of Fleischmann's yeast is poured on the surface. This yeast is what the flies eat. It grows freely in the sugar mixture. A disc of filter paper is placed over the surface of the food and a run-way of dry stiff white paper about 4×15 centimeters is placed vertically in a bottle. The flies are transferred from the stock bottle to a clean one by taking out the cotton stopper of both bottles, putting the mouths together and walking toward a window with the bottle in which the flies are to go held toward the light. They then pass from the old bottle to the new, the cotton plugs are again inserted, and a fresh colony is thus started. After the flies have sufficiently multiplied they are transferred to a clean dry bottle and a strip of glass about 3×15 centimeters is placed in the bottle. On this glass are put a number of small pieces of filter paper moistened by banana juice and tissue obtained by mashing up some pieces of banana and yeast with water. Flies are fond of this banana juice and proceed to lay their eggs on the moistened paper rather than on the dry glass. The glass strip is then removed, the papers lifted off with forceps, and placed in little boxes made up of celluloid obtained from old X-ray films. This is to prevent the eggs from drying. They may be exposed to radiation in these little boxes under any conditions which are desirable. The eggs, after exposure, are placed in a flat glass dish with a cover to prevent evaporation, a small quantity of the yeast-banana juice mixture being poured into the bottom of the dish to keep the paper moist. At the end of from 36 to 48 hours, depending somewhat on the temperature of the room, the papers with the eggs are placed on the stage of a microscope with a low power objective and the unhatched eggs and empty shells counted. This can be done with a squared eyepiece micrometer, or, if preferred, the eggs can be simply counted and pushed aside with a needle as enumerated. The empty shells represent the eggs which are hatched, the whole eggs those which have been killed. Elaborate counts of check series of eggs have shown 97 per cent to be fertile. The actual percentage of eggs hatching after radiation is therefore corrected by dividing it by 97.

In exposing the celluloid boxes to radiation they should be supported on a thin piece of gauze stretched over a wooden frame. There is, therefore, no back-scatter. If they were placed on any solid substance, such as a board or table, back-scatter would add an additional amount of radiation, depending upon the material and other condi-

TABLE I

ANALYSIS OF DATA TO SHOW THE AMOUNT OF VARIATION IN DOSAGE AS DETERMINED BY DROSOPHILA EGGS

Continuous current 0.154 e/sec. (determined by ionization tests)

Exposure	Percentage alive	Difference	Extremes	e/sec.	500 *R* given in
10 min. 92.4 *R*	87.0	−1.4			
	88.4	0.0			
	89.0	+0.6			
	87.9	−0.5			
	92.6	+4.2	H. 77 *R* / 600 sec.	0.128	65 min.
	92.4	+4.0			
	87.2	−1.2			
	91.2	+2.8			
	89.1	+0.7	L. 108 *R* / 600 sec.		
	82.5	−5.9		0.180	46 min.
	83.7	−4.7			
Average	88.4		93 *R* / 600 sec.	0.155	54 min.
Standard deviation		±3.06			
15 min. 138.6 *R*	63.6	−2.8			
	63.0	−3.4			
	68.0	+1.6			
	63.2	−3.2			
	63.1	−3.3	H. 135 *R* / 900 sec.	0.150	55 min.
	69.8	+3.4			
	69.4	+3.0			
	62.6	−3.8			
	67.5	+1.1	L. 150 *R* / 900 sec.	0.167	50 min.
	62.6	−3.8			
	63.8	−2.6			
Average	66.4		142 *R* / 900 sec.	0.158	53 min.
Standard deviation		±3.02			
20 min. 185 *R*	53.2	+4.9			
	48.1	0.0			
	41.7	−6.6			
	43.5	−4.6			
	46.7	−1.4	L. 207 *R* / 1200 sec.	0.172	48 min.
	53.4	+5.3			
	53.4	+5.3			
	43.4	−4.7			
	47.8	−0.3	H. 170 *R* / 1200 sec.	0.142	58 min.
	50.5	+2.4			
	49.4	+1.3			
Average	48.1		185 *R* / 1200 sec.	0.153	54 min.
Standard deviation		±4.00			
25 min. 231 *R*	37.8	+0.6			
	41.3	+4.1			
	35.4	−1.8			
	37.9	+0.7	L. 249 *R* / 1800 sec.	0.166	60 min.
	32.6	−4.6			
	41.6	+4.4			
	37.7	+0.5	208 *R* / 1800 sec.	0.139	50 min.
	38.8	+1.6			
	35.4	−1.8			
	38.2	+1.0			
Average	37.2		226 R / 1800 sec.	0.151	55 min.
Standard deviation		±2.62			

tions, so that the increased dose could not easily be allowed for.

In measuring radium, the tube of salt or emanation with the metal filter to be employed is dropped into a cylinder of hard rubber or bakelite four to six millimeters thick, to absorb the secondary radiation from the metal filter. The strips of paper on which the eggs have been deposited are wrapped about the cylinder, hence are equally distant from the radium cylinder. The whole is placed in a glass jar to prevent evaporation. About 50 per cent of the eggs are killed by 180 *R*-units and 90 per cent are killed by 500 *R*-units. As, however, these eggs are minute in size, this means there is no portal effect. Five hundred *R*-units usually gives a threshold erythema on a sensitive skin through a large portal, say, 10×10 or 20×20 centimeters. But it is best in testing X-ray machines to avoid scatter, because that phenomenon is to a certain extent a function of the wave length as well as that of the portal. The accuracy of the method is very considerable.

Fig. 1. Eggs of *Drosophila*. The scale in the middle of the picture is in tenth-millimeters.

Table I gives a record of observations made by a machine giving a completely smoothed continuous current. Attention should be called in speaking of continuous current to the fact that commercial machines using kenotrons for rectification do not give a completely smooth current, because they work on 60 cycle alternations, and to smooth this out would require an enormous condenser capacity, in itself occupying space considerably in excess of that required for the commercial apparatus. While such machines do give a considerably larger output of X-ray at the same milliamperage and same voltage through the tube.

as compared to the mechanical rectifying machines, they do not give as much as a completely smooth current which can be obtained from experimental apparatus running on 500 cycles with very large condenser capacity. The oscillograph tracings from this experimental machine show no irregularities in voltage. The first series on Table I are observations which have been made with 10-minute exposure. The dose was estimated by an open Duane chamber and was 92.4 *R*-units. The percentage of eggs hatching at each of the various observations is given. Each observation means, as a rule, from 200 to 400 eggs counted. The average will be seen to be 88.4 per cent, with a variation of only plus or minus a little over 3 per cent. The maximum deviation of a single observation lies between 77 and 108 *R*-units, or 65 as compared to 46 minutes exposure, from a dose of 500 *R*-units, while the total number average would be 54 minutes. Thus the fluctuation for a single observation is about 8 per cent plus or minus. Such a single observation, therefore, should not be used in calibrating a machine. But it will be noticed that these numbers are obtained from a short exposure which places the observations on the upper part of a curve shown in Figure 2. Here a very small difference in the percentage of eggs killed is a large difference in *R*-units. As we approach the central portion of the curve where 50 per cent of the eggs are killed, the difference becomes less, and without going into details the fluctuations of single observations lie between 55 and 50 minutes for 500 *R*-units, a difference of only 5 per cent plus or minus.

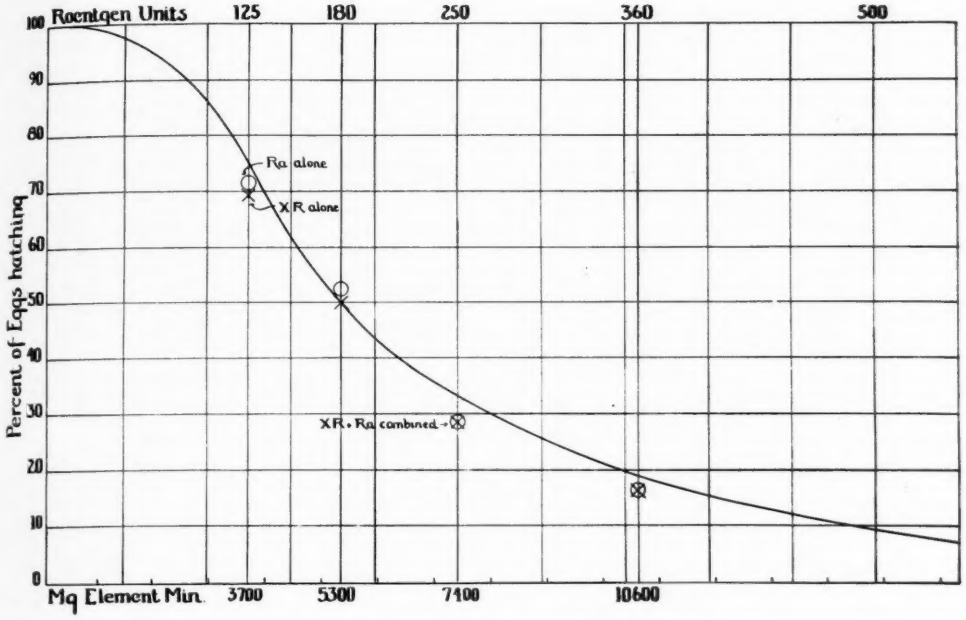

Fig. 2. Curve showing death rates from X-ray (crosses) and gamma rays (circles) and the results of combining equal doses of each. The curve is the standard X-ray and gamma ray curve obtained from many thousands of eggs and the observations are shown to fall very closely on this curve, thus proving that on *Drosophila* eggs two doses of different rays have the same effect as double the dose with either.

TABLE II

Mechanical rectification 0.154 e/sec. (determined by ionization tests)

Exposure	Percentage alive	Difference	Extremes	e/sec.	500 *R* given in
10 min.	83.0	−0.2	H. 100 *R* / 600 sec.	0.167	50 min.
	85.9	+2.7			
	83.4	+0.2			
	83.8	+0.6	L. 111 *R* / 600 sec.	0.185	45 min.
	81.5	−1.7			
Average	83.2		107 *R* / 600 sec.	0.170	49 min.
Standard deviation		±1.35			
20 min.	45.0	−3.8	L. 196 *R* / 1200 sec.	0.183	46 min.
	47.0	−1.8			
	43.4	−5.4	H. 175 *R* / 1200 sec.	0.146	57 min.
	52.3	+3.5			
Average	48.8		185 *R* / 1200 sec.	0.154	54 min.
Standard deviation		±3.84			

TABLE III

Mechanical rectification 0.154 e/sec. (determined by ionization tests)

Exposure	Percentage alive	Difference	Extremes	e/sec.	500 *R* given in
10 min.	78.2	−3.0	L. 119 *R* / 600 sec.	0.198	42 min.
	78.2	−3.0			
	87.3	+6.1			
	89.1	+7.9	H. 90 *R* / 600 sec.	0.150	55 min.
	87.4	+6.2			
Average	81.2		112 *R* / 600 sec.	0.187	45 min.
Standard deviation		±5.6			
20 min.	55.1	+2.8	H. 167 *R* / 1200 sec.	0.139	60 min.
	53.9	+1.6			
	51.5	−0.8			
	51.5	−0.8	L. 176 *R* / 1200 sec.	0.147	57 min.
	53.1	+0.8			
Average	52.3		175 *R* / 1200 sec.	0.146	57 min.
Standard deviation		±1.57			

The average dosage was 53 minutes for 500 *R*-units, so that the plus or minus fluctuations are only about two and a half minutes. That is, with such an egg experiment, a machine can be calibrated within 2.5 per cent plus or minus. This is much closer than an erythema and is just about as close as can be estimated by an ionization chamber without elaborate precautions.

The remainder of Table II shows similar observations, fluctuations being a little larger as we approach the higher death rates or the lower portion of the curve, so that in accurate machine testing a preliminary run should be made to find out about the 50 per cent point, or a considerable series of eggs exposed at times estimated as one-third to one-half a skin erythema. With a commercial machine using mechanical rectification, variations similar to those with a continuous current are seen, but they are also within the limits of variability of the skin erythema. The maximum deviation shown in Tables II and III will be seen in one case to be 46 and 57 minutes for 500 *R*-units, in another 42 and 55, and in still another 57 and 60. The latter observation, which is the most consistent, was obtained about the 50 per cent point. The machines on which this test has been made are not subject to excessive line surges and the filament current is controlled by Rieber stabilizers. There were no changes during these tests in the position of the resistance buttons or the autotransformer control, so that no doubt they were running as smoothly as any machine of this type will.

After alterations in the position of the rectifying disc, marked changes were noted, as has long been known, and after a recent re-wiring of the overhead connections an extraordinary increase in the output was observed, the amount of X-ray energy emitted being nearly twice that given by the machines before the wiring was done. The tubes were not changed and the control settings were all kept the same, but the voltage on the voltmeter across the primary of the transformer was about 10 per cent higher than before, and a number of patients received sharp erythemas. This led to testing the machines with *Drosophila* eggs, and a complete readjustment of the standard dosage was found to be necessary.

I think sufficient evidence has been brought forward to show that these small biological ionization chambers are useful in machine-testing. They, likewise, have permitted a variety of investigations which have been conducted in the past few years by Dr. Charles Packard in my laboratory. It was there first conclusively shown that with equal quantities of X-ray energy, measured with an open ionization chamber, the lethal effects were independent of the wave length.

The accuracy of these tests as compared with skin erythemas is evident when we realize that in some of these experiments twenty or thirty thousand eggs were counted. Each egg is equivalent to an erythema test, so that in a relatively short time a series of observations can be made which are statistically much sounder than those which can be obtained by a few skin erythemas, and the estimation of the effects is much easier, because every one acknowledges that it is difficult to judge the quality or the amount of an erythema. If we fall back on what may be called a "threshold erythema," even this is difficult to evaluate. With the flies' eggs the determining point is that the eggs either hatch or they do not, and an enormous series of observations can be obtained, the statistical accuracy of which is reflected in the small percentage of variations, as shown in Table I.

Unfortunately, it is impossible to measure radium in terms of roentgen units with the type of ionization chamber now in use, so that an accurate ionization comparison between radium and X-ray cannot be made.

Rather than assume that what is true of X-ray at a wave length of 0.6 Å. and at a wave length of 0.2 Å., is necessarily true of a wave length 0.01 Å., as has been done by certain makers of ionization chambers, it would appear to be safer to use these biological ionization chambers and test the results. When this was done it was found that the observations made upon radium could be exactly superimposed upon those made by X-rays of various wave lengths. The times of exposure vary enormously. To give 500 *R*-units with X-rays is a matter only of minutes, but to do it with 60 milligrams of highly filtered radium at a distance of six or seven millimeters may require eight hours. Nevertheless, as the curves show, the observed data superimpose, hence the laws governing the killing of flies' eggs with X-ray and radium are the same. Whether the erythema produced by equal amounts of unfiltered or long wave lengths of X-ray is the same as that produced by highly filtered gamma rays is another question yet unsettled, which may be left to the practitioner of radiotherapy. But the demonstration shows that the laws governing the killing of an individual cancer cell anywhere in the body either by X-ray or radium are the same.

The *Drosophila* eggs lent themselves to another interesting experiment, and that was to prove that the scatter and portal laws, as worked out on phantoms by ionization methods, were biologically correct. It was easy to check in a few weeks' work all of the charts which have been published as to back-scatter with different wave lengths of X-ray and different portals, and it would be equally easy to show depth doses. This problem is now under investigation. It is possible, from the Compton effect, that equivalent amounts of long wave lengths and short wave lengths of X-ray may not be equally effective after passing through materials of low atomic weight. As the eggs are killed, independent of wave length, this point can easily be settled.

Still another problem of radiation has been investigated. It has recently been said that the combination of gamma and roentgen rays, giving half a dose of each, requires a larger sum of radiation to produce an erythema than either alone.

Figure 2 and Table IV will show that in the case of flies' eggs two and two still equal four. In other words, a 50 per cent dose of gamma rays and a 50 per cent dose of X-rays make a 100 per cent lethal dose on the eggs of *Drosophila.*

Some other explanation, therefore, must be found for this curious variation in erythema doses, that is, if an erythema dose is a direct and quantitative response to some destructive effect on the epidermis and the neurovascular mechanism of the corium. If the clinical fact be true, it is a very important one, because it enables us to increase the clinical amount of radiation administered and still keep within legal limits, and, in consequence, the destruction of the tumor cells will be greatly enhanced by a combination that permits of higher dosage. But that a mixture of two types of radiation is more effective than either alone upon the cell itself as an individual organism, I very much doubt.

Finally, the use of this extremely convenient and easily obtainable biological material might be suggested as a means of calibrating in roentgen units, the present greatest need of the radiologist. It constitutes a simple and rugged ionization chamber which can be used by a person not a professor of physics—an apparatus whose price will be within the financial limits of the ordinary practitioner of radiotherapy. That is the great desideratum at the present time. It is necessary, still, to rely upon some rule-of-thumb method to determine erythema doses, and one is hopelessly in the dark as to the output of X-ray machines after re-

TABLE IV

Test of biological effect of approximately equal doses of X-ray and the gamma rays of radium. Note that a correction is made in computing the percentage alive for the presence of 3 per cent of non-fertile eggs.

X-ray only			Radium only			X-ray and radium		
125 *R*-units			3700 mg. element minutes					
Alive	Dead	Percentage alive	Alive	Dead	Percentage alive	Alive	Dead	Percentage alive
114	56	69.1	124	58	71.7	273	709	28.5
Expected from standard chart		75.0	Expected		75.0	Expected		33.0

180 *R*-units			5300 mg. element minutes			X-ray and radium		
78	86	49.4	40	57	42.5	134	645	17.7
62	62	51.5	84	109	44.8	73	345	18.0
148	156	50.3	110	88	57.2	161	934	15.2
			397	364	53.8			
288	304	50.2	631	618	52.1	368	1924	16.6
Expected		50.0			50.0			19.0

pairs have been made upon them or whenever a new Coolidge tube replaces the old. Instrument-makers are prepared to furnish at a reasonable figure sensitive and accurate galvanometers which will stand considerable handling. The necessary potential can be obtained from the electric light circuit, or, if that is alternating, from a B battery or rectifier. Certainly the alarming increase of damage suits for alleged overdosage might be diminished if weekly checks were made on therapeutic apparatus. All of the simpler forms of ionization chambers at present must be corrected for wave lengths. This correction can be simply and accurately made in roentgen units by the employment of *Drosophila* eggs.

Also, anyone using radium can determine the erythema dose without difficulty for a standard pack or other set-up of a group of tubes, or for any individual needle or tube, if necessary. Once this is obtained for a given amount, filter, and distance, a table can be prepared to save the necessity for future calculations..

I do not for one moment presume to suggest that the practising radiologist should test his own machines with *Drosophila* eggs; rather, that dealers in radiological supplies should furnish a cheap ionization chamber calibrated in *R*-units with the eggs. But any one who wishes to spend a week profitably during the summer, when professional duties are light, can catch a few flies and make in-

teresting observations on the irregularities of his machine and in determining surface and depth doses.

CONCLUSIONS

1. The present desideratum in radiotherapy is a cheap, rugged ionization apparatus which can be manipulated either by a physician who has not been trained in the use of elaborate physical apparatus, or even by a competent technician.

2. Such an instrument should be calibrated by the makers in *R*-units, and this can most easily be done by the use of *Drosophila* eggs.

DISCUSSION

Dr. H. J. Ullmann (Santa Barbara, Calif.): First, I wish to emphasize the practical value of this method. I had used the Solomon iontoquantimeter and had had my machine calibrated with a standard instrument by Dr. Newell. This was made by the physicist at Stanford University. I had checked my meter readings very carefully, and was still a little afraid of my dose, although I had had no accidents. After visiting Dr. Wood and seeing the work of Dr. Packard, I immediately tried the egg method, and found that what I called my 100 per cent dose was essentially the same as that which Dr. Wood called his sub-erythema dose, a dose that would kill approximately 90 per cent of *Drosophila* eggs. I believe, however, that it would be wise, or almost a necessity, to have an ionization chamber under the tubes, read by a galvanometer, because in any treatment the tube output may fall without a change in the meter readings. I found out early, while working with the Solomon iontoquantimeter, that such a chamber will tell whether or not the tube is maintaining its output at any moment during the treatment. The cost of such a chamber would not be over a hundred dollars. The galvanometer costs fifty or fifty-two dollars. The chamber you can make yourself. The charging outfit may be a B battery eliminator which costs from eighteen to twenty-five dollars, and the rest is simply wire connections. This is the method used by Dr. Failla.

Another important point which Dr. Wood did not stress is that in calibrating by this method the eggs must be placed on the patient or on a paraffin block, because if you make your calibrations with the eggs in air, you are going to overdose your patient. The dose that will kill these eggs is the erythema or sub-erythema dose, and there is a distinct back-scattering with high voltage into the skin; therefore, the reading must be taken on a paraffin block or on the patient. It is really very simple to do. You should read Dr. Packard's article as to the best way to raise the flies. It is simple to get them; go down to the fruit room in your hospital and put a piece of old banana in an open milk bottle and leave it twenty-four hours and you will have a nice collection of both flies and eggs,—they multiply better than guinea pigs. They will not always lay when you want them to. One must have a reasonably warm room or they will not always lay when wanted.

I found important use for them in checking my new thirty milliampere outfit against the five. It is stated that the output is considerably less than six times the output of a five milliampere tube, and yet I found that those who had been using the thirty were giving the same number of milliampere minutes. I found that a factor of six to one, the ratio between five and thirty, would kill the *Drosophila* eggs. I believe that this was due to the fact that the intensity is greater than one to four, and therefore the effect is greater; at least it checked so on *Drosophila* eggs. I was thankful that I had not increased my dosage.

Dr. Wilhelm Stenstrom (Minneapolis): Dr. Wood has presented a very interesting method. It is evident that the sta-

tistical method of measuring anything is good so long as large numbers are used, and if Dr. Wood's method is applied at other places, you have to remember that large numbers have to be used and that several measurements should be taken. We must remember that this is a method of transferring the standard dose from one place to another. We are still relying upon the ionization chamber as the fundamental instrument for standardizing the dose or for keeping the dose standardized somewhere in the country. Dr. Wood also evidently agrees upon that. The ionization chamber gives a more accurate value. If *Drosophila* eggs are used, we must keep in mind that there are certain precautions which should be taken. It is possible that several factors may influence the effect on the eggs, and I hope that Dr. Wood will point out if he has found any such factors: for instance, the age of the eggs—whether or not there is any temperature limit, or if light would influence the eggs. For example, some one starting to use them may put them in sunlight without thinking of it. I just mention these points because I think we ought to be very careful when we use biological material for measurements.

Dr. Wood pointed out that the different strains of *Drosophila* eggs have the same sensitivity. It may very well happen that a strain may be found that has a different sensitivity: we can never feel absolutely sure about that. Of course, we have to be very careful when we assume that the laws that govern the *Drosophila* eggs also govern human tissues. We may make an assumption that that is the case, but we should be very careful in drawing such conclusions.

I think there are other means to measure doses which may be as simple and at least as accurate. I am thinking of chemical methods. Hugo Fricke has recently radiated ferrous sulphate and measured the dose that was necessary to change it to ferric sulphate. It was a rather simple method, though the measurement of the ferrous sulphate change required some delicate instruments and some accurate work. I think that there are other chemical changes which might be measured in a much simpler way, and I shall refer to that in a paper I am presenting later. I agree with both Dr. Wood and Dr. Ullmann, that it is a very good idea to have a simple ionization chamber with a low sensitivity galvanometer in order to keep the dose standardized after we once have the standard.

Dr. Wood (closing): I am very glad to have Dr. Ullmann bring out several practical points I did not have time to do. In answer to the queries about conditions of working which Dr. Stenstrom made—the eggs do well below forty-five to fifty F. If the room is very cold, some of them are apt to die off, and also much heat or sunlight will kill a certain number, so control checks are always done with the sets which are exposed.

The question of the fertility of the eggs is an interesting one: the numbers which normally hatch are about 97 per cent as based on thousands of observations; so a small correction is made to adjust that error. Beyond keeping the eggs slightly moist and avoiding excessive temperature and keeping the flies at room temperature, there are no difficulties.

As regards the breed of flies, there are no differences between laboratory homozygous strains and wild ones. Prof. Holthusen has verified our results with flies from Hamburg. Details about the methods of breeding will be found in my paper and need not be gone into here, but the point is that it is difficult for the man in the street to buy a simple, rugged, and correct reading ionization chamber. *Drosophila* eggs give correct readings and can be used by the manufacturer to calibrate his chambers in biological units.

INTERPRETATION OF THE SEMINAL VESICULOGRAM[1]

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AT the start, your essayist is anxious that it be very clearly understood that he has never been nor is he now a so-called "enthusiast" over vas injection.

Excepting the subject of ureteral stricture, there is no question of an urological aspect that seems farther removed from a reasonable solution than that of the clinical value of medication of the seminal tract by injection of the vas.

Throughout all of the literature on this subject, considerable side-interest is attached to the question of possible harm done by the procedure. Opinions, based upon clinical observation, are almost unanimously to the effect that sterility is an exceedingly uncommon result of the procedure. These clinical observations seem to be borne out by the weight of such experimentation as has been done on the subject.

The remaining factor, that of its radiologic aspects, has received surprisingly little attention in the general literature on the subject of vas injection. Belfield, in 1913 (1), first called attention to the possibility of visualization of the vesicles by filling the cavity with some shadow-casting solution. Since then nearly every essay or text on the subject has referred to this factor, but, with very minor exceptions, the consideration has been that of technic of injection and of photography rather than of interpretation of the film itself.

Through the courtesy of the Superintendent and Resident Staff of the Milwaukee County Hospital, the opportunity of study of a fairly large series of cases of vas injection presented itself, an opportunity of which advantage has been taken. In addition to this, unlimited X-ray facilities were graciously offered and numerous retakes were made besides the original films. To make possible the presentation of that material, the services of the Art Department of Marquette University School of Medicine were graciously offered.

Throughout this work, comprising a few over two hundred injections, study has been made both of the clinical results following the injection and of the diagnostic information resulting from the procedure. The radiological aspects of this study, alone, are herewith presented. In all cases, the procedure consisted of open dissection and needle-puncture of the bared vas. In each instance, the solution used for injection was that of thymol iodide in cod liver oil, as suggested by Belfield and Rolnick (2).

As will be evident later in this presentation, in some instances there was little more to recommend the procedure than an earnest desire for knowledge regarding it. A series of normal vesiculograms was obtained by injection of the vas in cases in which it was exposed for some ordinary procedure, such as castration for tumor or operation for hydrocele. Another series of cases of acute gonorrhea complicated by epididymitis was injected, both on the involved and the uninvolved side. A rather large series of injections was done on cases of rheumatism, acute and chronic, associated with and apparently caused by acute or chronic seminal vesiculitis. In still another group, injection was done where the clinical picture was that of long-standing or recurring gonorrhea, with chronic prostatic and vesicular infection and, in many instances, recurring

[1]Read before the North-central Branch of the American Urological Association at Columbus, Ohio, Nov. 2, 1928.

epididymitis. A small series of cases of tuberculous epididymo-vesiculitis is included.

This presentation would be incomplete without reference to the possible harm resulting from vas injection. In this series there have been occasional instances of "back-fire" epididymitis, always, however, mild in degree and of short duration. In the distinct majority of cases, and in spite of everything that was done to obviate it, within one to three days after injection, an area of soreness, swelling, and induration developed within and around the cord at the site of puncture of the vas. This usually was of the size of a pea or small marble, bothered the patient but a few days, and after weeks or months remained only as a palpable node of scar tissue. Occasionally this area of swelling grew in size, softened, was opened, or opened itself, to drain a watery matter heavily charged with the iodide in a flaky or precipitate-like form.

The rather common occurrence of this induration at the site of puncture has raised the question of possible sterility following the procedure. In only seventeen cases, where bilateral injection had been done, was it possible to study condom specimens collected weeks or months after injection. Three of these cases had been injected twice; one, three times. In one of the cases, an asperma of many years had been known to exist before injection. In the other sixteen, sperms of normal activity and morphology were found. The possibility of occlusion of the vas on one or the other side, could, of course, not be denied in any of these cases. A total of thirteen vasa were injected the second time in from three to twenty-one weeks after the first injection. In each instance the second puncture was done at a site below that of the first, and in each case the lumen at the point of previous puncture was proven to be open and free, both by passage of a strand of silkworm gut and by flow of the solution. Similar patency was noted in the case on which a bilateral injection was made the third time. In one instance of known sterility of long standing, at the time of reinjection, that segment of each cord involved in the first injection was resected. On serial section, perfect patency of the lumen was noted in each vas, though in each a heavy node of organized scar was seen embedding the cord. From all of this there has been nothing in this series of cases, more than the bead of dense scar so often felt at the site of puncture, that would indicate the danger of sterility following the procedure.

For the sake of description, that part of the seminal tract commonly visualized by the X-ray after vas injection may be divided into: (1) the vas; (2) the ampulla of the vas; (3) the body of the vesicle; (4) the neck of the vesicle, and (5) the ejaculatory duct.

From 2 to 3 c.c. of solution is required to completely fill each seminal tract. Even the lesser of these amounts is usually sufficient to completely fill the vesicle and result in some overflow. The excess, of course, forces the weaker of the two urethral sphincters and regurgitates into the bladder. The shadow produced by the overflow has the value of insuring patency of the ejaculatory duct as well as complete filling of the vesicle. It has the disadvantage of obscuring the shadow of the vesicle unless care is exerted to insure the proper direction of the ray. With the target centered over the pubis, as in the first of these films, the overflow shadow is liable to obscure the vesiculogram. With the target centered over the umbilicus, so that the ray is directed downward in the line of axis of the bony pelvis, the overflow shadow is seen well above the vesiculogram.

If a patient is able to void immediately after the injection, most or all of this over-

flow shadow is eliminated. In those cases in which general anesthesia has been given, or in which the patient, for any other cause, is unable to void for several hours after injection, considerable drainage of the vesicle is often found to have occurred. The vesiculogram then shows incomplete filling, and, therefore, poor visualization of the vesicles. In Figure 2, taken after voiding and five hours after injection, the solution, escaping from the vesicle, is seen filling the prostatic urethra, and the vesicles are seen only partly filled.

To avoid the confusion of the overflow shadow, catheterization was done, but it was usually found that the heavier oil did not come away easily, and that remaining, especially with the bladder collapsed, had a great tendency to overlay the shadow of the vesicle and render the film useless.

The normal vesiculogram, like the normal pyelogram, varies to some extent in different men. It has certain factors, however, which are quite constant. The vas deferens is often visualized from the point of puncture, coursing up over the pubic ramus and down around the lateral wall of the pelvis, usually curling upon itself before leaving the lateral pelvic wall and turning mesially, where it soon begins to dilate slightly and coil in very fine rolls to form its ampulla. At times small, bottle-necked, diverticulum-like buds are seen springing from the ampulla, particularly near its union with the neck of the vesicle.

The vesicle itself presents a rather complex arrangement of delicate tubule-like or cellule-like cavities, having a tendency to converge in a central indefinite canal, which narrows into a short duct-like neck, promptly joining its vas to form the ejaculatory duct.

The ejaculatory duct, when seen in normal men, appears as a fine, thread-like canal almost parallel with its mate, but converging slightly for a distance of about one-half to two-thirds inch, and coursing downward and forward to its opening into the urethra.

Normal vesicles have a startling similarity to each other. The cellules of each vesicle simulate each other in size and type, and though those near the tips of the vesicles are at times larger than those near the neck, difference of consequence in their size and shape is not seen in normal vesicles. Though the cavities of the vesicle of one man may be larger and more capacious than those of another, in the individual normal vesicle there is a constant delicateness and fineness in the detail of its cavity.

The first of these vesiculograms is beautifully normal. In the second, though the cellules at the tip of each vesicle are more capacious than those of the body, the perfect symmetry of the two vesicles would seem to indicate them to be normal.

It has been quite definitely established by the report of several observers that the solution injected by vas puncture gradually leaks from the vesicles, so that normally

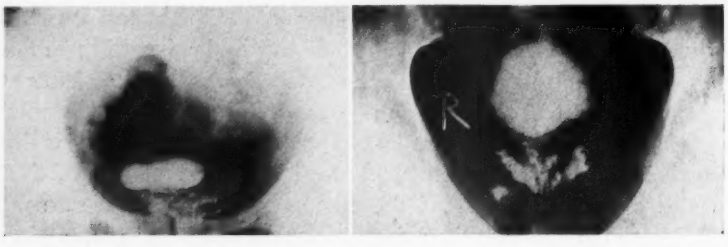

Fig. 1. *Left:* Target over the symphysis—overflow bladder shadow obscuring the vesiculogram. *Right:* Target over the umbilicus—overflow bladder shadow well above the vesiculogram.

they empty within two to five days. Observations in this series verify this finding. In those instances, however, where definite pathology existed, it was not uncommon to find a very material lengthening in this emptying time. It seems altogether possible that this emptying time might fairly be taken as an index to the absence or presence of obstruction at the vesical neck or in the ejaculatory duct.

The upper series of pictures in Figure 5 are of a case of chronic, bilateral seminal vesiculitis, with frequently recurring acute gonorrheal urethritis over a period of seven years. Though the second film shows the usual partial emptying in four days, the third film, taken nine days later, shows a 50 per cent retention. The lower series is taken of a case of chronic gonorrhea complicated not only by prostatitis and vesiculitis but by arthritis. Fifty per cent of the drug is seen to be retained after the first week, and 25 per cent of the drug still remains after three weeks. The retention in each case is definitely beyond normal limits.

Fig. 2. Partial emptying of vesicles in five hours.

In many respects Figure 6 shows the most remarkable vesiculogram in this series. This

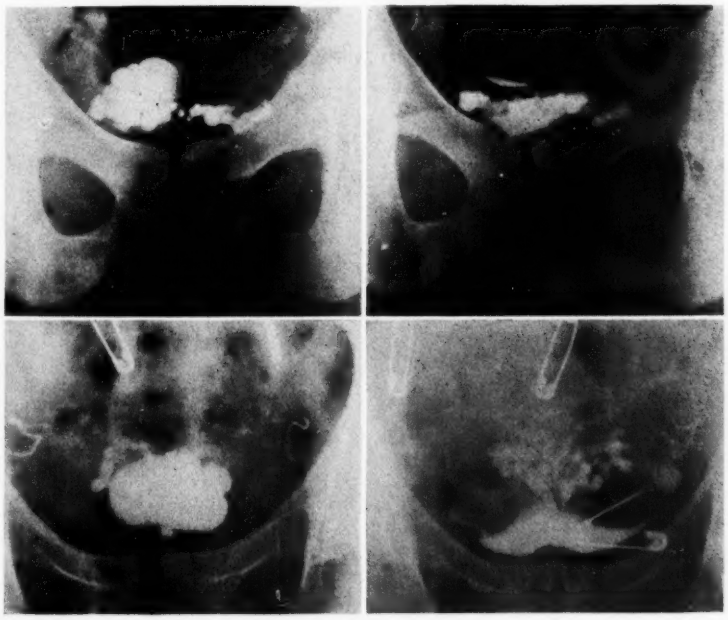

Fig. 3. Top row and bottom row each shows a case before and after catheterization.

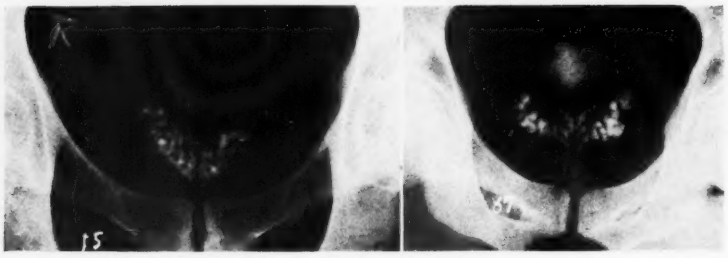

Fig. 4. Normal vesicles.

case occurred early in this work and at a time when different amounts of the oil were being injected to determine that most suitable. Five c.c. was injected on the right and 10 c.c. on the left. From the enormous distention of each vesicle, together with the absence of bladder overflow, it is evident that there is complete occlusion of each ejaculatory duct. The three-week recheck shows much of the drug still present and traces could be seen on a film taken four months later.

Occlusion of the vas is not an uncommon finding and seems to occur by preference at the coil of the vas just before leaving the lateral pelvic wall. The first of these two cases (Fig. 7) was one of acute left gonorrheal epididymitis. By forced injection, the vas ruptured at the point of occlusion (indicated by the arrow) and the solution extravasated down the lateral pelvic wall to the area marked by the letter "O." The second case was one of bilateral tuberculous epididymitis in which both vesicles were palpable as contracted beaded cords. The right vas is occluded at the site where it coils before leaving the lateral pelvic wall. The left vesicle will be noted as a small, cramped cavity—a finding to be referred to later.

In reviewing the vesiculograms included in this series, it soon became evident that deviations from the normal vesicular capacity and contour consisted both of con-

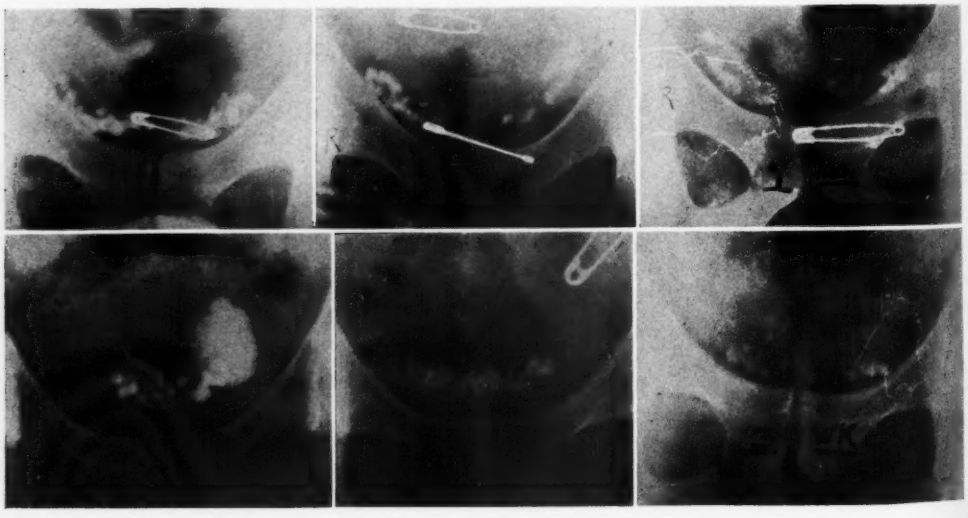

Fig. 5. *Top row:* Nine-day retention. *Bottom row:* Three-week retention.

Fig. 6. Occlusion of ejaculatory ducts, with three-week and four-month retention.

traction and of dilatation. Furthermore, that in the case of dilatations, there seemed to be two separate and distinct general types of dilatation: that involving part or all of the vesicle itself and that involving the ejaculatory duct and ampulla of the vas.

By comparing the clinical findings with the two types of dilatation, it seemed that in those cases presenting dilatations of part or all of the vesicle, the condition was one of relatively recent, subacute inflammation. It seems altogether likely that such dilatation represents muscular atony of the vesicular wall, the result of inflammation.

By further comparison between the clinical history and the changes seen in the vesiculogram, it seems apparent that this atonic dilatation tends to involve the vesical neck when epididymitis is an outstanding part of the clinical picture, whereas such dilatation, when found to involve the main body of the vesicle, is quite commonly associated with a clinical picture dominated largely by associated arthritic manifestations.

The cases presented in Figure 8 are representative of atonic dilatation of the vesical neck. In these cases the clinical history was that of subacute gonorrheal seminal vesiculitis of from three to five months' duration, and with rectal findings in which the vesicles were palpated as normal. As has been suggested, this type of atonic dilatation, involving more the vesical neck, seemed to be rather uniformly associated with epididymitis. In the first two cases there had been bilateral epididymitis early in the disease; in the third case, only on the right side.

The cases shown in Figure 9 are representative of dilatation of the body of the

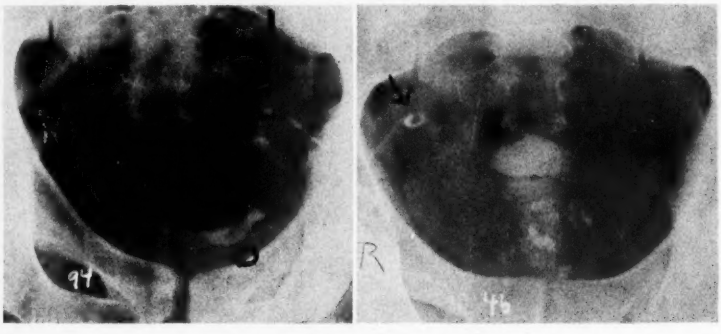

Fig. 7. Occlusion of vas deferens.

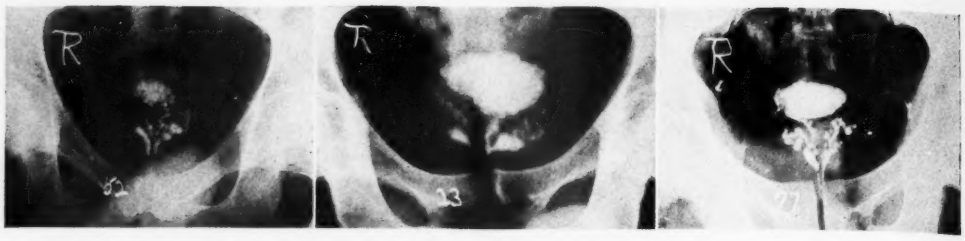

Fig. 8. Atonic dilatation of the neck of the vesicle, a finding commonly noted in cases of chronic gonorrheal vesiculitis in which epididymitis has occurred.

vesicle. In all, the clinical history was that of subacute or chronic gonorrheal seminal vesiculitis of from seven to twelve months' duration, associated with chronic, low-grade, polyarticular arthritis. In what appeared to be definite contrast with the preceding group, these cases seemed to be unassociated with earlier epididymitis. Here, as before, the vesicles felt *per rectum* were either normal or of the "soft distended" type.

Dilatations involving the ampulla of the vas and ejaculatory ducts, but not the vesicle proper, were met with less frequently. Furthermore, they seemed rather uniformly associated with a clinical history of very long-standing chronic and recurring gonorrhea, often with repeated attacks of epididymitis. The question arises as to whether this dilatation does not differ materially from that previously referred to; whether, in fact, it is not due to actual stricture of the ejaculatory duct with hydrostatic dilatation above, rather than muscular atony as suggested before.

Both of the cases shown in Figure 10 were of very long-standing, more or less active and recurring gonorrhea, with repeated attacks of epididymitis. In Case No. 38 both the right ampulla and the ejaculatory duct are seen to be enormously dilated above the point where they enter the urethra. In Case No. 39 the ampulla is definitely dilated above a point near its junction with the vesical neck. The extreme contraction of the vesicle on the involved side might reasonably be expected to indicate extensive

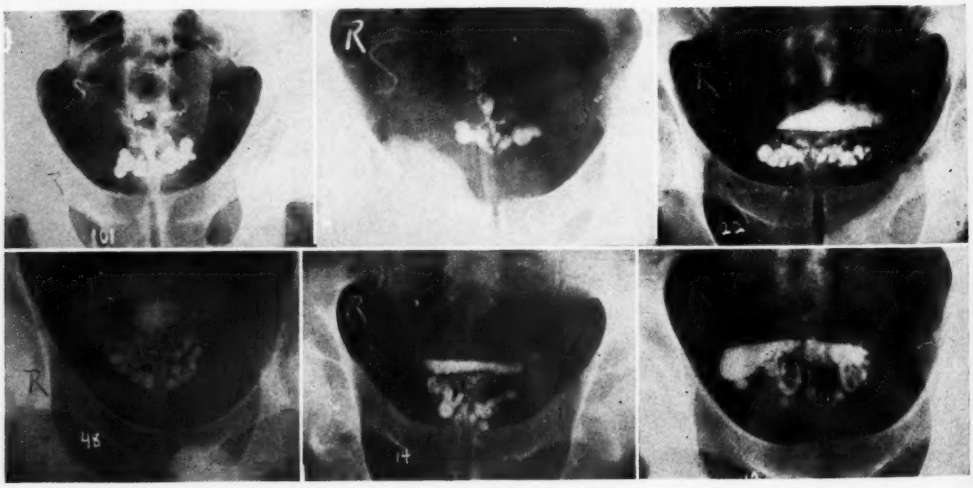

Fig. 9. Atonic dilatation of the body of the vesicle, commonly noted in cases of chronic gonorrheal vesiculitis associated with arthritis.

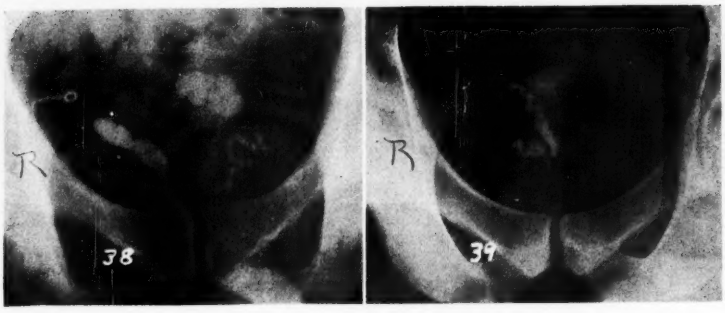

Fig. 10. Sausage-like dilatation of the ampulla of the vas and (in Case No. 38) of the ejaculatory duct, a very long-standing process and probably due to true stricture of the ejaculatory duct.

sclerosis of the vesical wall from long-standing inflammation, a phase about to be considered.

Contraction of the vesical shadow, like that of dilatation, was met with in a fairly large percentage of these vesiculograms. Also, like that of dilatation, it seems to occur in two distinct and separate ways.

In those cases of recent acute gonorrhea with unilateral epididymitis, it was found that, by comparison, the vesicle of the involved side was considerably reduced in general size. This appeared so constantly that, from the films of all these cases, like the three presented in Figure 11, the side upon which the epididymitis had occurred could be told without error by this comparative diminution in size. Of these three cases, the acute epididymitis was on the left in Case No. 57, on the right in Case No. 45, and on the left again in Case No. 6.

In explanation of this, the thought suggests itself that this diminution in apparent size of the vesicles of the involved side merely represents acute inflammatory swelling of the mucosa and wall of the vesicle. With such as the case, more or less return of the vesical shadow to normal upon recovery of the inflammation might naturally be expected.

This, in fact, does occur and is illustrated in Case No. 10 (Fig. 12). This particular patient had had subacute gonorrhea for several months, with a recent exacerbation complicated by a moderate right epididymitis together with a typical gonorrheal arthritis, monarticular in type, and involving the right hip. The left vesicle shows some atonic dilatation at its tip; the right presents the apparent diminution in cavity so consistently seen in recent epididymitis. The second film was taken five weeks later, after reinjection, and, incidentally, following a clinical recovery, possibly as startling as was observed in any of this series of cases. This second film presents both vesicles practically

Fig. 11. Decrease in the size of the vesicle from acute inflammatory swelling of its walls, a finding constantly associated with acute gonorrheal epididymitis.

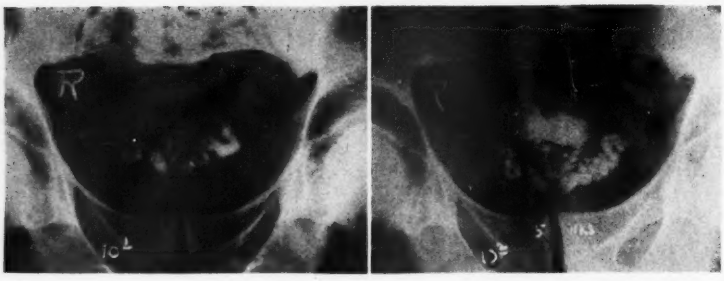

Fig. 12. *Left:* Usual findings in acute gonorrheal epididymo-vesiculitis. *Right:* Same patient, re-injected five weeks later and after clinical recovery.

normal in appearance. It may be worth stating here that changes of any consequence in the outline of the vesicle, as revealed by reinjection of the vas, were met with very infrequently, and then, as in this case, only when the inflammatory process in the vesicle had been of comparatively short duration.

The second type of diminution in the size of the vesicle seems to be that of a true sclerotic contraction of the vesical wall, the result of long-standing inflammation.

Each of these four patients (Fig. 13) had had chronic, more or less active, gonorrhea, complicated by frequently recurring attacks of epididymitis on either side, and lasting over periods varying from two to five years. The right vesicle in Case No. 44, and both vesicles in Case No. 63, present an extreme degree of cicatricial contraction, almost to the point of obliteration. Both sides in Case No. 58, and especially the left side in Case No. 109, present the same changes though to a lesser degree.

As would be expected, this type of vesiculogram is always found in patients with long-standing chronic gonorrheal seminal

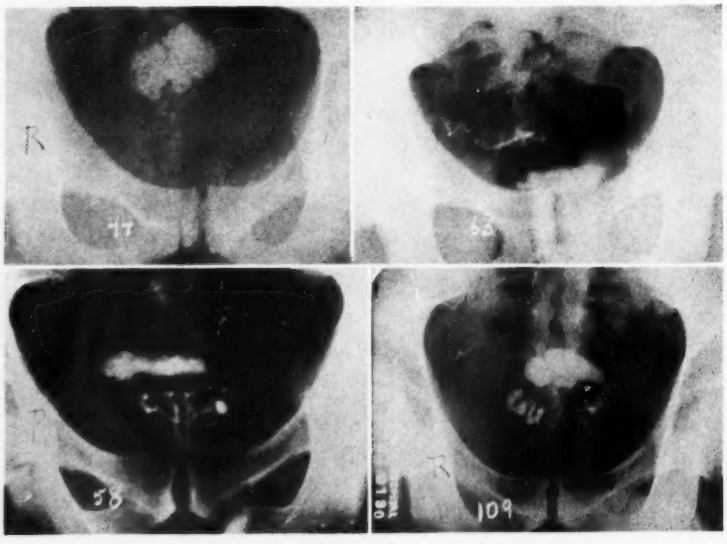

Fig. 13. Sclerosis and contraction of the vesicle resulting from long-standing inflammation.

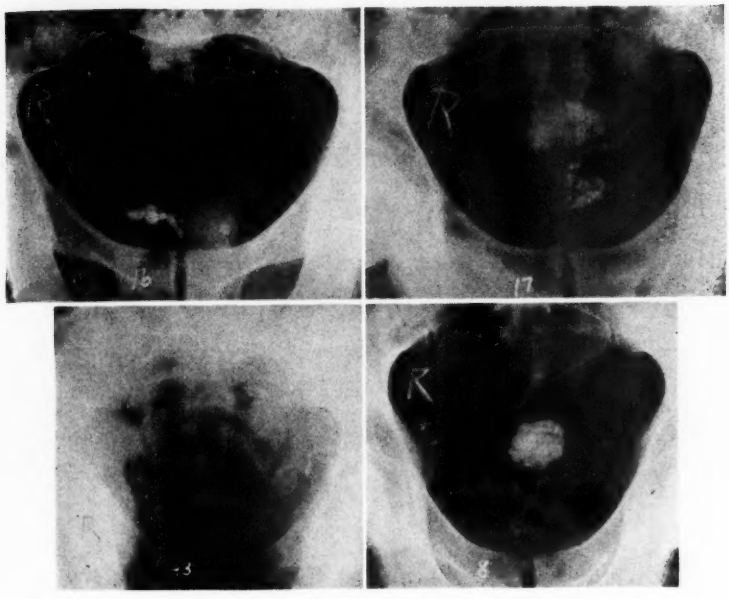

Fig. 14. Changes in the vesiculogram seen in genital tuberculosis.

vesiculitis, and in whom the vesicles are felt *per rectum* as small, hardened, rope-like cords.

In a rather small series of cases, in which either epididymectomy or orchidectomy was being done for tuberculosis, the exposed vas was injected. The changes in the outline of the vesicle presented nothing radically different from those seen in the non-tuberculous inflammatory lesions. Case No. 16 (Fig. 14) was one of early tuberculosis of the right epididymis, with normal urine and without change in the palpatory findings *per rectum*. The vesiculogram differs little, if any, from normal. Case No. 17, also an early one, was found to have some thickening of the left vesical wall *per rectum*. Though comparison with the opposite vesicle cannot be made, it appears that there is some cramping of the vesicular cavity on the involved side. Case No. 43, one of chronic bilateral genital tuberculosis, shows the right vesicle generally contracted, whereas the left vesicle is not apparent though the vas can be seen throughout its course. Case No. 8, one of chronic right genital tuberculosis, shows the vesicle to be nearly obliterated. This vesicle, as was true on both sides in Case No. 43, could be felt *per rectum* as a hard, contracted, beaded cord on the back bladder wall.

Lowsley and Kirwin (3) have published a vesiculogram showing a cavity the size and shape of a small almond, connected with the seminal vesicle, and visualized by solution injected through the ejaculatory duct. This was interpreted as being an abscess of the seminal vesicle and the diagnosis was proved at operation.

In the case shown in Figure 15, when the first film was taken, a small, round shadow about 1 cm. in diameter was seen overlying the right seminal vesicle and apparently caused by a globule of oil separated from the main mass producing the overflow shadow. The second film, taken after voiding, though showing the overflow shadow to be completely gone, still presents the

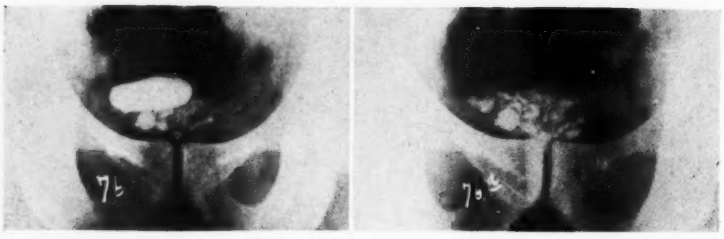

Fig. 15. Abscess of the seminal vesicle (before and after voiding).

Fig. 16. Anatomical anomalies visualized by vas injection.

rounded shadow. Stereoscopic films proved this in close apposition and apparently connected with the cavity of the right seminal vesicle. The case was one of chronic, bilateral epididymitis, suspected of being tuberculous. Whether this shadow was that of an abscess communicating with the seminal vesicle was never proven. It, however, closely simulates the picture of proven abscess of the vesicle referred to above.

It was not surprising in this series to meet with anatomical anomalies.

Case No. 81 (Fig. 16) shows the right vas deferens and ampulla carried over to the extreme left of the pelvis in company with the normally placed left vas. Cases No. 7 and No. 1 present the not uncommon finding of duplication of the vesicle.

SUMMARY

A series of vesiculograms, visualizing a few over two hundred seminal vesicles, have been reviewed and compared with the clinical findings. This includes normals as well as cases of acute and chronic gonorrheal vesiculitis, chronic non-gonorrheal vesiculitis, and tuberculous vesiculitis.

The vesiculogram is constant and does not vary from a characteristic normal in normal men, nor does it vary from time to time in the same individual so long as the condition of the vesicle remains the same.

Dilatation of the seminal vesicle, in part or in whole, occurs commonly in cases of chronic vesiculitis of several months' duration. This dilatation is thought to be atonic in nature and is not permanent if the inflammation subsides.

Dilatation of the ampulla of the vas or of the ejaculatory duct occurs, the result of organic stricture, and, when present to any degree, remains permanently.

Diminution in the cavity of the vesicle is a constant finding in acute gonorrheal epididymo-vesiculitis, and is thought to be due to inflammatory swelling of the vesical wall. This change is temporary and disappears with recovery from the disease.

Contraction of the wall of the vesicle, with extreme decrease in the cavity, even

to the point of obliteration, occurs as the result of long-standing inflammation. This change in the vesiculogram is seen in chronic vesiculitis, either of gonorrheal or tuberculous origin.

The vesiculogram reveals abscess of the seminal vesicle, when present, and communication with its cavity.

Obstructions of the seminal tract, as well as anomalies of the vas and seminal vesicle, are revealed in the vesiculogram.

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THE SPHINCTERS OF THE COLON

By R. BALLI,

Professor of Radiology, Royal University of Pavia, Italy

ANATOMISTS generally describe but two sphincters in the colon: the ileocolic, which I would call the sphincter of Varolius, and the internal sphincter below the rectal ampulla. It is true there are references in Quain, Huntington, Tillaux, Rose, Barles and Beirne to muscular thickenings in the colonic wall, but, in general, anatomists have ascribed no particular importance to these, and have not dignified them by calling them sphincters.

The anatomic researches of Rutherford on the so-called ileocolic valve of Varolius have demonstrated that this valve has a characteristic structure; namely, that it is more than a valve with the simple function of passively regulating the passage of the intestinal contents and checking the backflow, but that it is to be considered a complex, active, regulating mechanism.

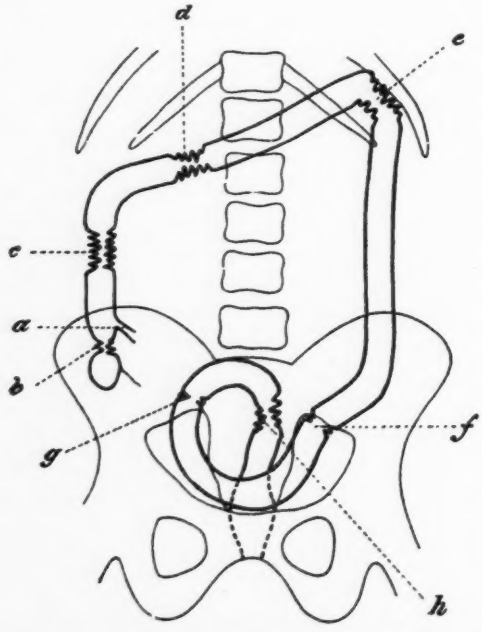

Fig. 1. Diagram illustrating the location of the different sphincters: (*a*) sphincter of Varolius; (*b*) sphincter of Busi; (*c*) sphincter of Hirsch; (*d*) sphincter of Cannon; (*e*) sphincter of Payr; (*f*) sphincter of Balli; (*g*) accessory sphincter of Moutier (Rossi); (*h*) sphincter of Moutier.

RADIOLOGY

The radiologic viewpoint may be described briefly (Fig. 1), as follows:

(*a*) *Ileocolic sphincter of Varolius* (Fig. 2). The credit for having accurately studied this sphincter goes to an Italian radiologist, Busi, and his school. "The terminal end of the ileum," he says, "directed more or less obliquely from below upward toward the large intestine, opens into it at an angle more or less acute. Just where it passes into the large intestine, there is a rounded ampullaform dilation, one to two centimeters in diameter, which is divided from the rest of the ileum by two opposing incisuræ. It terminates in the large intestine, as a triangular shadow with a more or less sharp apex." This shadow, which lies almost perpendicular to the longitudinal axis of the large intestine, represents the space contained in the so-called ileocolic valve. Its apex corresponds to the central orifice of a mammillary eminence protruding into the large intestine. The dimensions of this shadow vary in the same subject at different times during the examination, and it assumes characteristic contours in pathological cases. Radiographically, the central orifice of the mammillary eminence must be interpreted as being open when the triangular or oval image spreads out, or blends with that of the large intestine, and closed when the apex of the shadow is faint but sharp. The contour corresponds to "a bird's beak forma-

tion," in Busi's apt expression. Fluoroscopically, especially in left lateral decubitus, cylindrical fragments are passed from the ileum into the large intestine, through peristaltic action of the terminal end of the ileum. The entry into the cecum may, however, cease entirely, even if the terminal ileum be full and in active peristalsis. All this corroborates the presence of an ileocolic sphincter exercising active control. Busi thinks, and I agree with him, that the function of the sphincter of Varolius can be aided, modified, and sometimes supplanted by the contraction of other contiguous parts of the cecum, especially by a sphincter which he thinks he has isolated, and by the terminal ileum.

Fig. 2. The sphincter of Varolius in contraction.

Fig. 3. Spasm of the sphincter of Busi in a case of appendicitis.

(*b*) *Sphincter of Busi,* or colicocecal sphincter (Fig. 3), is described, from the radiologic point of view, as a sphincter between the cecum and the ascending colon below the ileocecal orifice, which gives the impression that the cecum seems divided from the ascending colon by an incisura or by a circular deep sulcus, and in some cases "it seems as if attached to the ascending colon by a slender stem, like an apple on its branch." According to Busi, the colicocecal sphincter does not usually go into contraction immediately upon arrival of ingested food in the large intestine, but only some time afterwards, demonstrating its anatomic independence of the ileocolic sphincter. For some time it may be visualized, but it soon relaxes to make room for another sphincter situated in the ascending colon—the sphincter of Hirsch. In cases of dilation of the cecum, I have seen what appears to be a spastic area at this site. Busi has also demonstrated this in cases with Jackson's membrane and in cases of appendicitis.

It is not uncommon to find in the cecum contemporaneously with the sphincter of Busi, and in relation to its lower border, an incisura which Montanari thinks represents the contraction of a tenia (the anterior). This, as Busi has noted, may extend to the cecum and ascending colon as a more or less transparent band, longitudinally disposed and showing through the haustra. I believe that the action of the sphincter of Busi and of this small band are important in the progress of content passing from the cecum to the colon.

(*c*) *The sphincter of Hirsch* (Fig. 4). We come across this sphincter regularly in the proximal segment of the ascending colon, namely, above the cecum. This sphincter, which has been the object of diligent and detailed study by Hirsch, is called by the author "cecocolic." I prefer using the name "cecocolic" for the sphincter of Busi. Sometimes it may be found near the hepatic flexure, and it may assume the shape of a sphincteric tract (Montanari). Busi, in several cases of filling the colon with enema, after evacuation of the enema, has noted that the whole large intestine empties into the sphincter of Hirsch. Proximal to this point the cecum and ascending colon remain well filled.

According to Hirsch, the cecocolic segment shows an active retention of the intestinal contents until the cecal digestion and the reabsorption are complete. He thinks that the contraction and relaxation of this portion of the intestine, analogously to what happens in the opening and closing of the pylorus, is regulated by chemical action.

Spasm of the sphincter of Hirsch has been noted in appendicular lesions (George and Leonard, Döhner, Gottheiner and others). Hirsch has found it in cases of ulcerative tuberculosis of the ileum, of adhesions of the ileum, of colitis, of Jackson's membrane of the ascending colon, in cases of tumor of the duodenum, etc. I have found such spasm in a case of ileocecal tuberculoma and in a case of appendicitis and right adnexal adhesions, besides appendicular lesions. If the spasm is persistent, there is stasis and dilation of the cecum similar to that produced by an infiltrative process of the ascending colon.

Fig. 4. Spasm of the sphincter of Hirsch in a case of appendicitis with extensive adnexal adhesions.

Fig. 5. Spasm of the sphincter of Cannon in a case of aorto-mesocolic stenosis.

(d) Sphincter of Cannon (Figs. 5, 6, 7, and 8). Cannon, in 1902, and Bohm, later, have shown the presence of a sphincter in the transverse colon about the junction of the first and third portions, or, in other words, between the proximal part of the colon and the distal part. It extends over an area of about a centimeter, and is usually distinct. In the normal state of tonic contraction it becomes visible as a sudden narrowing of the haustral formations.

Even in the same individual, the sphincter

of Cannon may vary in its position, either to the right or to the left, due, according to Busi, "to the state of relaxation or contraction of the small bands." But the interaction between the superior and inferior mesenteric plexuses and their variable distribution in the transverse colon may account for this. I have found the spasm of the sphincter either isolated or associated with the sphincter of Hirsch in some cases of cholecystitis, in functional stenosis of the third portion of the duodenum, constipation, appendicitis with adnexal adhesions, and in digestive vagotonia.

(e) Sphincter of Payr and Strauss (Fig. 9). The contracture described by Payr and Strauss is situated at the left flexure of the colon. I have found spasm of this area in a case of mega- and dolichocolon.

(f) Sphincter of colon-sigmoideo (Fig. 10). The colosigmoidal sphincter demonstrated by me is produced by a tonic or spastic contraction of the colon at its junction with the sigmoid. I have met the spasm of this sphincter in cases of constipation (Fig. 13).

(g) Sphincter of Moutier (Fig. 11). Moutier has described a sphincter in the terminal colon, or, to be more exact, between the sigmoid and the rectum. This sphincter, I believe, corresponds anatomically to the one mentioned by Beirne as being located at the level of the third sacral vertebra. I have observed the spasm of this sphincter in cases of constipation. Rossi has described an additional spastic area observed in cases of habitual constipation in the adult, but particularly in the child, at the level of the middle portion of the sigmoid.

Fig. 6. Spasm of the sphincter of Cannon in a case of cholelithiasis.

Fig. 7. Spasm of the sphincters of Cannon and Hirsch in a case of habitual constipation.

SURGICAL ASPECT

Considerable light is thrown on the complicated mechanism of habitual constipation by the X-ray examination. This discloses the modifications in peristalsis and tone and the disco-ordinated action of the sphincters which modify and alter the normal action of the colon. The sphincters are the points of commencement of peristaltic and antiperistaltic waves.

The antiperistaltic movements produce retrograde transportation of the colonic contents. This retrograde movement is sometimes slow, sometimes rapid. This back-flow may have its starting point at Cannon's sphincter, or from the sphincter of Hirsch, or from the sigmoid. In these retrograde movements, true antiperistaltic waves may flow from Cannon's sphincter to

the cecum, or from the sigmoid to the Hirsch sphincter. The above mentioned facts are not unknown in surgical practice. Hartmann, during an operation of ileosigmoidostomy on a patient with a fistula of the ascending colon, saw the formed fecal mass regurgitate through the fistula, whereas feces excreted from the natural anus were liquid. Croves and Hall, in two cases of ileosigmoidostomy, were obliged, fifteen months after, to intervene with a right hemicolectomy because the feces, by a retrograde ascension through the excluded anus, lost the liquid part by absorption and formed stercoraceous concretions of hard consistency and sometimes in great quantity in the excluded segments. Similar cases are reported by F. De-Quervainda, A. Lane, Stierlin, Gietz, Beck, Mattioli, Korte, Pieri, Pazzi and others.

This antiperistaltic reflex following ileosigmoidostomy is thus associated with the existence and function of the sigmorectal sphincters below the anastomotic mouth, and with the presence above of an empty colon deprived of normal activity for want of physiologic stimulus (Rossi).

Lane ascribed importance, not always confirmed, to the efficiency of his mesocolonic contractile ligament in checking, partially at least, such phenomena. But no author mentions any opposing resistance of muscular action by the colon. The question now arises, in reference to these phenomena—for instance, in an ileosigmoidostomy—whether they constitute a contra-indication to this operation—a constant danger or at least an inconvenience.

Fig. 8. Spasm of the sphincters of Hirsch and Cannon.

Fig. 9. Spasm of the sphincter of Payr in a case of colicocolon (after administration of barium enema). The spasm is slightly below the angle.

Mattioli insists that the preference be given, in suitable cases, to the operation of ileotransversostomy. His reason is not fear of the activity of these contractile zones, but that ileotransversostomy is immune from the post-operative complications which follow unilateral exclusion of the whole large intestine (colon). In diarrheal flatulence, the feces, passing directly from the ileum into the transverse colon, find themselves in a segment of colon where the function of the reabsorption of liquids predominates, whereas the physiologic peristalsis provoked by the feces themselves opposes successfully the damage of antiperistalsis.

To some surgeons the existence of these retrograde movements, which may be eliminated by operation, have a great importance. In the opinion of others the contractile zones of the colon do not possess such definite, constant, and distinct functions as to overrule any projected operative procedure. There is, however, no justification for underestimating their importance in the normal functioning of the colon. Our studies tend to confirm in this regard the common processes of adaptation to the profound modifications produced by surgery in various parts of the gastro-intestinal tract, by which a compensatory system is established so as to permit the reassumption of function, not identical, but at least approximating the normal physiological régime.

In this regard both Chiarolanza's studies on the histologic changes in the excluded colon and the radiologic work of Spangaro on his own cases, operated on for total colectomy, are particularly important. The authors show that the terminal part of the ileum, in these cases, increases in diameter and that motor functions are assumed similar to those observed in the normal colon. It is analogous to the perfect functional adaptation which one observes in the great majority of the cases operated on for ileocecal resection or pyloric resection, by entero-anastomosis and gastro-entero-anastomosis.

Fig. 10. Spasm of the colon-sigmoidal sphincter in a case of habitual constipation.

Fig. 11. Spasm of the sphincter of Moutier in a case of habitual constipation.

Having considered the sphincters of the colon from the anatomic, radiologic, and clinical points of view, we will now discuss the essential nature of the sphincters. Have these sphincters, thus radiologically demonstrable, any anatomic substrata? Does the colon at the points where the sphincters have been described, have a structure identical with parts of the colon without sphincters? These researches were made on the colons of feti, newborn infants, and adults.

HISTOLOGY

1. *Sphincter of Varolius* (Fig. 12). According to the majority of anatomists, this sphincter, seen from the ileum, presents

Fig. 12. Anterior view of ileocecal area in a six-month fetus, to demonstrate the sphincter of Varolius.

something like a cuneiform cavity which retracts together towards the colon. Seen from the side of the large intestine it looks like an oblong protuberance composed of two lips, folds, or valves, which extend forwards and backwards forming the frenula valvulæ or retinacula. This valve is considered as the result of a partial insertion of the ileum into the colon. The serous tunic and the longitudinal layer of the muscular tunic are absent. Besides, Toldt believed he had proven that this invagination included the deep layer of the longitudinal fibers of the ileum and the colon.

These anatomic conceptions have been completely modified by Rutherford. He, in 1914, proved by anatomic studies of animals killed suddenly and on human corpses a few hours after death, that the ileocecal valve in this fresh state has an appearance quite different from that described by the anatomists. Besides, the valve presents itself as a spherical eminence with a diameter of 1.8 cm., smooth and shining, which contracts into the colon from ½ to 1 cm. with blunt cavity and perforated in its center by an orifice of varying shape either directed transversely with lobulated lips or concave downwards, or circular or like a star. Neither the two valves nor the correlative frenula exist. Such arrangements were noted by Rutherford also in the living body in 1905 in a case of cecal fistula.

The anatomic structure of the mammillary eminence, according to the author (Fig. 13), consists of two layers of circular and longitudinal fibers which are peculiar to the ileum and which penetrate the cecum, and a layer of circular fibers. These form a powerful muscular sphincter. Externally overlying these is a third layer of circular fibers; that is the muscular circular layer of the mammillary eminence. At the apex (top) of the valvulæ the three mentioned circular layers come together, forming a common layer of circular fibers.

My researches not only confirm histologically those of Rutherford, but add details, which, according to my opinion, are very valuable from the physiologic point of view. Indeed, from the innermost layer of the terminal ileum, longitudinal fibers radiate, terminating in the circular layer of the mammillary eminence. There are muscular fibers running obliquely—almost ring-like; there are other fibers, also in oblique direction, which part from the innermost layer of the longitudinal fibers of the colon and terminate in the circular layer of the ascending colon, in the cecum, and also in the circular layer of the mammillary eminence.

From these observations it is logical to deduce that the function of the last part of the ileum is not only dominated by circular fibers, as Rutherford asserts, but also by oblique fibers coming from the ileum as well as from the large intestine. These fibers should, it seems to me, begin to function after the relaxation of the circular fibers of the sphincter, and the content would be ejected through the sphincters not only be-

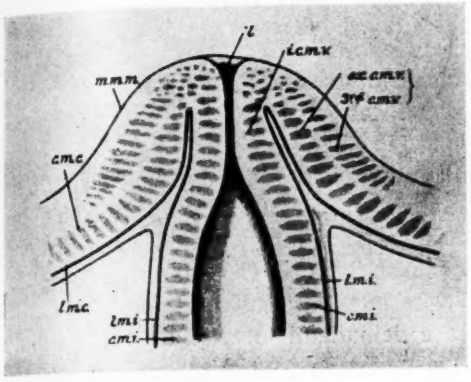

Fig. 13. Diagrammatic drawing demonstrating the sphincter of Varolius in vertical section, after Rutherford: *1* = lumen; *mmm* = mucous membrane of the mammillary eminence; *cmi* = circular muscular coat of the ileum; *lmi* = longitudinal muscular coat of the ileum; *lmc* = longitudinal muscular coat of the large intestine; *cmc* = circular muscular layer of the large intestine; *icmv* = internal circular muscular layer of mammillary eminence; *excmv* = external circular muscular layer of mammillary eminence; *3rd cmv* = third layer of the circular muscular fibers of the mammillary eminence. This is considered as a separate layer, but it is probably a doubling of the external circular layer of the valve itself.

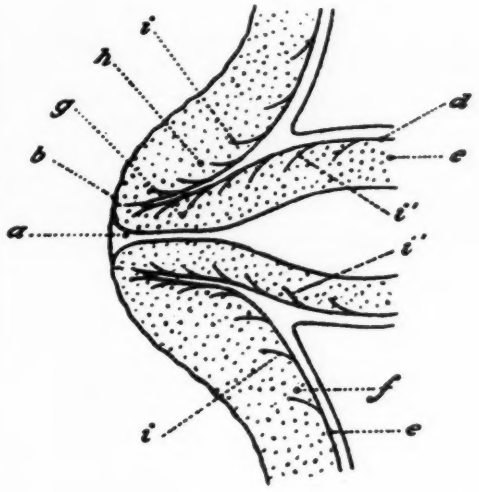

Fig. 14. Sketch illustrating the structure of the sphincter of Varolius, in vertical section: (*a*) lumen of the mammillary eminence; (*b*) mucous membrane of the mammillary eminence; (*c*) circular muscular layer of the ileum; (*d*) longitudinal muscular layer of the ileum; (*e*) longitudinal muscular layer of the cecum; (*f*) circular muscular layer of the cecum; (*g*) circular muscular layer of the mammillary eminence; (*h*) circular muscular layer of the ascending colon; (*i*) oblique fibers originating from longitudinal fibers of the cecum and ascending colon, which enter the circular layer of these two segments, and of the mammillary eminence; (*i' i''*) oblique fibers originating in the innermost layer of the longitudinal fibers of the ileum, which enter the circular layer of the ileum and of the mammillary eminence.

cause of intestinal peristalsis, but also as a result of contraction of these fibers. Such phenomena were found by Rutherford and myself. The function of the valve is exactly similar to that of a sphincter like the pylorus, and regulates the passing of the chyme from ileum to cecum.

Such a conception is confirmed by the anatomic studies of Luschka and of Keith (thickening of the muscular layer of the ascending colon in the cat and in man), by the physiologic researches of Elliot (contraction of the valve after stimulation of the splanchnic nerves and relaxation of the ileum on section of the nerves), and by the clinical researches of Busi and Rossi in cases of marked dilatation of the ascending colon, with stasis not accompanied by insufficiency. The embryological studies of Beati on the sphincter of Varolius prove also, although indirectly, the important function of this part.

Before entering upon the histologic study of the other sphincters, I believe it necessary to refer briefly to the anatomic structure of the colon as it is now described. The anatomists describe the large intestine as consisting of four coats: the serous, the muscular, the cellular, and the mucous.

The muscular coat interests us in this particular study. The disposition of longitudinal and circular fibers does not present special differences throughout the colon.

The longitudinal fibers, united in two or three distinct bands (according to Lowitz Brites) in the cecum and colon and in the zone existing between bundles, produce a continuous stratum (layer) of longitudinal fibers, more or less complete. This begins

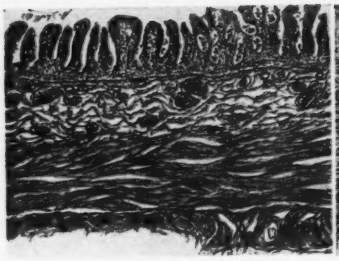

Fig. 15. Transverse section of a portion of the colon where there is no sphincter.

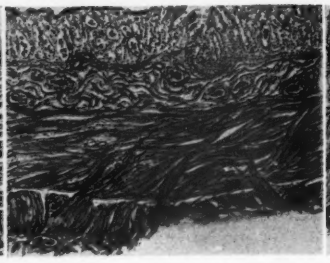

Fig. 16. Transverse section of the sphincter of Busi.

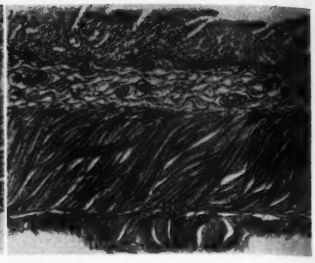

Fig. 17. Transverse section of the sphincter of Hirsch.

to widen at the level of the ileopelvic colon, to form, in relation to the rectum, a separate layer of fibers which occupies the whole intestinal periphery, thinner over the lateral surface than over the anterior and posterior faces of the rectum itself. In the rectum, according to Laimar, some longitudinal fibers change their directions and continue with the circular fibers, and these circular fibers act in an analogous fashion with the longitudinal fibers. The circular fibers of the large intestine, according to the anatomic description, form a continuous band which involves the whole circumference, and which is prolonged throughout the whole length. This layer of fibers is very thin, thinner than the longitudinal fibers, and the fibers which constitute it are very pale. Also, the circular layers in the rectum, as well as the longitudinal fibers, have an arrangement which should be understood. The thickness of this layer in relation to the rectal ampulla is less than in other regions. Beneath the rectal ampulla it thickens to the anal orifice, where it forms a muscular ring—the internal sphincter. According to Brites, the two muscular layers are not adherent to each other, as in the case of the ileum, where it is possible to make a mechanical separation, but they are united to each other through anastomosing bundles. Finally, at the level of the passage of the vessels of the internal coat, small circular bundles

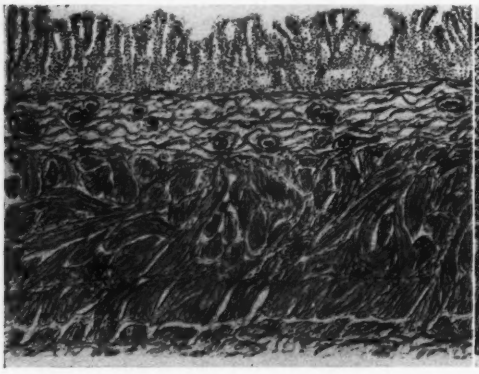

Fig. 18. Longitudinal section of the sphincter of Hirsch.

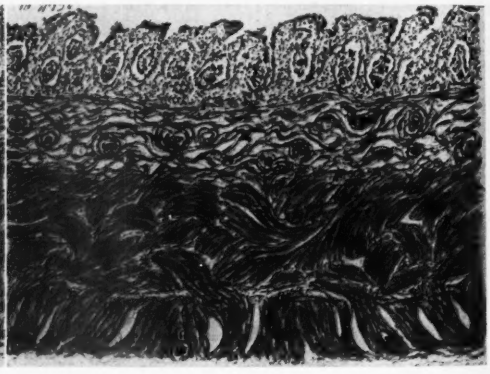

Fig. 19. Transverse section of the sphincter of Moutier.

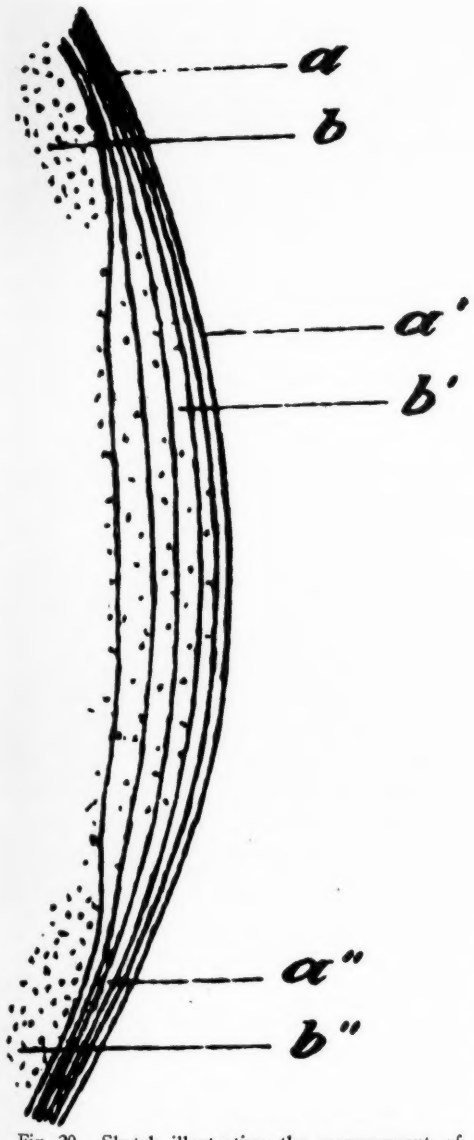

Fig. 20. Sketch illustrating the arrangement of the circular and longitudinal fibers at the level of the sphincter of Moutier (vertical section): (*a a''*) circular fibers of the jejunum above and below the sphincter; (*b b''*) longitudinal fibers of the jejunum above and below the sphincter; (*a' b'*) distribution of the longitudinal and circular fibers in relation to the sphincter.

change direction to follow the above mentioned vessels to the external coat and further.

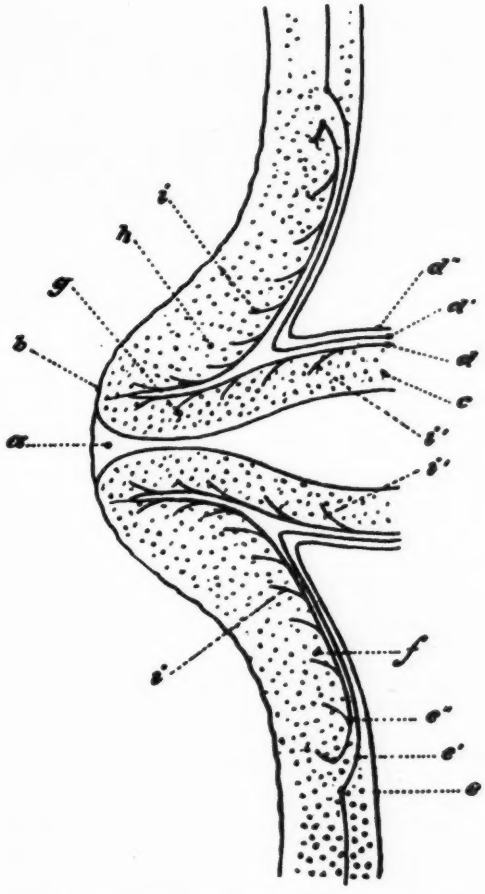

Fig. 21. Drawing showing the arrangement of the longitudinal terminal fibers of the ileum in the cecum and in the ascending colon (vertical section): (*a, b, c, d, e, f, g, h, i, i'*) as in Figure 20; (*d, d', d''*) longitudinal muscular covering of the ileum, which continues, respectively, in the cecum in *e*, with direct longitudinal fibers (independent of the longitudinal fibers of the small bands proper) and which disappear below in the cecum and above in the ascending colon at different levels, in *e'* and *e''*, which cross the circular layer of the cecum and the ascending colon where they terminate.

Let us see now what happens in the sphincteric zones.

2. *Sphincter of Busi* (Fig. 16). Histologic studies of the zone corresponding to this sphincter disclose an increase of longitudinal and circular fibers over the normal, and the passage of fibers from the longitudinal layer to the circular layer. This is par-

ticularly marked in close proximity to the site of the sphincter of Varolius. The vessels run from the longitudinal layer to the fibers of the circular layer and end in the submucosa and mucosa.

3. *Sphincter of Hirsch* (Figs. 17, 18).

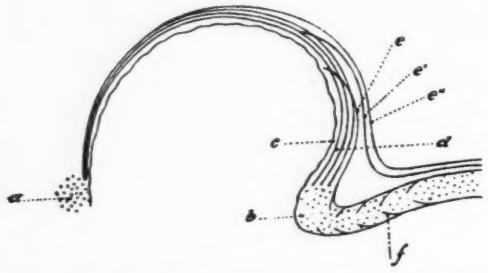

Fig. 22. Sketch illustrating the arrangement of the fibers of the ileum in the colon in a transverse section traced at the level of the sphincter of Varolius; (*a*) postero-lateral band; (*b*) mammillary eminence; (*c*) mucosa; (*d*) circular fibers proper of the large intestine; (*e, e', e''*) circular fibers of the large intestine coming from the longitudinal fibers of the ileum; these form, in part, the circular layer of the large intestine, and become lost among these fibers; (*f*) oblique fibers (see longitudinal section).

In relation to the zone which is the seat of the sphincter of Hirsch, there is a much more marked increase in thickness of the circular fibers and more fusion of longitudinal and circular fibers than is found about the sphincter of Busi. Besides this, there are in the circular layer fibers interlacing in bands of musculature. The disposition of the vessels corresponds to that described above.

4. There is no change from the usual structure of the colon at the site of the *sphincter of Cannon.*[1]

5. *Sphincter of Payr.*

6. *Sphincter of colon sigmoidic of Balli.*

7. Accessory *sphincter of Moutier (Rossi).*

8. *Sphincter of Moutier* (Figs. 19, 20). In the zone of the sphincter of Moutier, one notes interlaced bands, as above, thicker than Bartes has found in the sigmoidal inlet. The vessels are distributed as in the other sphincters.

Because of this arrangement of interlacing longitudinal fibers with circular fibers, and because the mesenteric plexus follows the course of the vessels, the cellular interspace where the ganglia of the plexuses are located remains always wrapped with a muscular reticulum. This structure is confirmed by Reiss, and is in accordance with my researches on the sphincters. The autonomous centers of the intestine are found near or in the sphincters, centers which are united to each other by nerve fibers, thus causing the synergy of the contraction of the muscular tunic (layer) and the possibility of a reciprocal influence for the most various stimuli.

In the light of these anatomic studies, only four of these sphincters present definite muscular markings which justify the term "sphincters" in the true sense of the word (Varolius, Busi, Hirsch, Moutier). The others, in my opinion, are functional sphincters, without anatomic substrata.

In view of these facts, the question arises whether the increase of longitudinal and circulary fibers in the sphincters of Busi and Hirsch is an autonomous increase or arises from other elements.

I have studied the arrangement of the terminal longitudinal and circular fibers of the ileum in serial section in the fetus and in the newborn, and I have found that in longitudinal sections of the cecum, ileum, and ascending colon, most of the longitudinal fibers of the ileum are reflected over the cecum and ascending colon, partly with the longitudinal fibers of the cecum and ascend-

[1] Tillaux cites a particular reaction of the muscular actions of the transverse colon, which is worthy of mention: "I shall relate," he writes, "a singular anatomic fact, from which I would not at present make any deductions. Often, in the transverse colon I found a sharp constriction of a portion 8 to 10 cm. long. Distal to this was a portion of small caliber intestine. This narrowness was more than half in proportion to the perimeter. Many times, I encountered the same narrowness in the fetus with the intestine full of meconium. This stricture disappears completely after insufflation, without leaving any trace in the intestine or in the intestinal walls, to explain this fact."

ing colon, and partly with the layer of circulary fibers of these two portions (cecum and colon), and terminate there (Fig. 21).

In the transverse section of the colon at the level of the Varolian sphincter, the longitudinal fibers of the ileum partly encircle the colon at the periphery and partly enter the circular layer of the colon, ending there.

Such an arrangement, which indicates the presence of other non-autonomous fibers from other parts, seems to me to be of great significance. In fact, the intimate relation between the muscles of the ileum and those of the ascending colon (cecum), together with the special disposition of the so-called "Auerbach's Collar," may explain the normal or pathologic correlation between the action of the cecum and colon, and between the ileum and the cecum, giving this region importance, anatomically, physiologically, and clinically.

A SOURCE OF DANGER IN THE USE OF VERTICAL ROENTGENOSCOPES AND TUBE STANDS

By JOHN R. CARTY, M.D., Clinic of the Cornell University Medical College, NEW YORK

THE electrical and radiation hazards inherent in the use of X-ray apparatus have been well considered. Accidents from these sources are comparatively rare now. There is, however, a danger which is not generally appreciated, incidental to the use of necessarily heavy weights in vertical roentgenoscopes and tube stands. A recent accident during the use of a vertical roentgenoscope has focused our attention upon this subject. The problem is essentially a mechanical one, and although having the benefit of expert advice we feel a natural hesitancy in invading a somewhat unfamiliar field. In the last analysis, however, the responsibility for the safety of the patient remains with the roentgenologist and we feel justified in presenting the subject from his point of view.

Twelve physicians and others who constantly use X-ray apparatus were asked whether they had encountered accidents involving the parting of counter-weight cables, the falling of lead cones, etc. To our surprise seven reported that they had experienced such occurrences, the majority of which involved the parting of counter-weight cables. Agents of five leading makes of X-ray apparatus were then questioned as to the frequency of accidents of this type. We were able to obtain definite figures in only one case, but the other replies indicated that they had all experienced trouble from this source. One agent stated that his company "used to have a lot of trouble from the breaking of cable," another said that "no tube carrier had ever fallen on a patient," while another recalled two cases which had occurred recently.

The relatively high incidence which we report is probably coincidental, but, if taken in connection with the replies from the manufacturers' agents, should be considered significant. If a more extended search were made there is no doubt that many instances of heavy weights falling could be unearthed. Such accidents are, we believe, more frequent than is generally thought.

A search of the literature of the last three years showed comparatively little on the subject. This may be due, in part, to the fact that the Continental trend of design is towards lighter apparatus, particularly for radiography. The self-protecting tube has done away with the necessity for heavy, clumsy protection from primary radiation. The case reported in the *British Journal of Radiology* for August, 1927, and the communication regarding it in the same journal a few months later are of special interest.

Inspection of the leading makes of vertical roentgenoscopes and tube stands shows a considerable diversity of design of the counter-balancing mechanism. Multiple strand steel cables are employed in the majority, although two firms use chain. One manufacturer uses bronze cable instead of steel. In only one instance are twin cables installed,—this with a heavy stereoscopic chest plate changer. The cables are usually attached to the tube carrier by being bent around a shank and the end fastened to the cable by means of a bolt. Occasionally two bolts are used, if an exceptionally heavy weight is to be supported. In one instance the end is fastened by means of wire wrapped tightly around the cable.

Considerable variation in size (diameter) of the pulley wheels has been noted. This variation apparently has no relation to the amount of weight to be supported. Means

of locking the tube carriers on the tube stands are employed by all.

Vertical roentgenoscopes present two distinct designs. In the first type the screen forms an integral part of the tube carrier, being attached to the latter by means of an arm. With this design, if the cable snaps, both the carrier and fluorescent screen will fall to the floor. Sufficient clearance from the floor is allowed so that injury to the patient's foot would be impossible. If, however, a child is being examined while raised from the floor, the screen might injure him. In the second type the screen is not an integral part of the tube carrier but is attached to a counter-weight by a system of cables and pulleys, the counter-weight, in turn, being connected with the tube carrier by a cable. The screen and carrier move simultaneously, but if the cable between the carrier and counter-weight parts, the carrier falls, while the screen ascends with great violence. If the patient's chin happens to extend in front of the screen a severe or even fatal injury may be received.

Questions regarding the design and construction of counter-balancing mechanism of vertical roentgenoscopes and tube stands were asked of an eminent authority on mechanical engineering, who made the following statement: "With adequate inspection of a well designed apparatus the only failures to be expected are those due to overload and those of defective material. Visual inspection would not usually reveal defective material or incipient failure from this cause." He further states that, "The size of a pulley used with a cable or chain is of great importance, both in respect to wear and failure due to defective or overloaded material. There is no reason why properly designed cable or chain should not give satisfactory service. A cable is far less likely to fail through defects than a chain, hence I would consider a cable safer than a chain of the same estimated strength. Except that bronze cables are more flexible than those of steel, I would consider steel preferable to any other material for either cable or chain."

From the point of view of the mechanical engineer, counter-balancing mechanism of X-ray apparatus should conform to certain standards of design and should be regularly inspected by a competent mechanic, while periodical replacement of cable should be made to insure against possible defects of material. Only one of the concerns interviewed emphasized periodical inspection of apparatus. Others admitted that such an inspection might avoid accidents of this type.

Case 1.—While a physician was using a vertical roentgenoscope, suddenly the tube carrier fell, and at the same time the fluorescent screen ascended with great violence, narrowly missing the patient's chin. The primary circuit remained closed for a few seconds (probably the patient stepped on the foot switch) and the fluorescent screen made contact with the high tension circuit, in some manner never satisfactorily explained. The patient received a moderate shock, but fortunately no great harm was done. On investigation it was found that the cable connecting the tube carrier with the counter-weight had parted at a point just below where it went over the pulley. This cable had been visually inspected about six days previous to the accident, and there had been at that time no evidence of wear or defect.

The cable of a roentgenoscope is subject to more wear than a tube stand if there is no means of locking the tube carrier, thus causing the load to be constant.

Cases 2 to 5.—These were similar to the one reported above and occurred in the same type of roentgenoscope. No electrical shocks were received, however, and the cables had not been inspected. In one instance the cable parted during the night, but in the others patients were in place. Details of injury

were difficult to obtain, but one patient received a severe nervous shock from which she was months in recovering.

Case 6.—A doctor was adjusting a therapy tube carrier, with a patient on the table beneath. He had unlocked the carrier, but with one hand was firmly grasping it. The cable parted, but the physician caught the carrier before it reached the patient, thus avoiding injury. It is not known whether the cable had been previously inspected. This brings up an important point: if the carrier is unlocked above a patient, one hand should always firmly grasp the carrier.

Case 7.—While adjusting a tube carrier of a vertical tube stand the cable parted. The patient received an injury, the extent of which was not ascertained. In this case there had been no inspection of the cable and the latter was found to be badly worn in several places.

Case 8.—A radiograph tube stand fell: fortunately, the patient was not in position. Inspection revealed that the bolt fastening the end of the cable to the tube carrier had become loose, allowing the end of the cable to slip. This occurred on a new outfit. These bolts should be carefully watched, especially when a new cable has been installed.

Case 9.—While irradiating a patient in the lithotomy position for pruritus vulvæ, the lower half of the protective lead-lined box of a therapy tube carrier fell, carrying with it the tube and narrowly missing the patient. The current was turned off before the patient made contact with the high tension line, and no shock was received. Investigation revealed that the safety of this apparatus, when being used in other than the horizontal position, depended upon a small wing nut. It was found that this nut would not remain tight.

CONCLUSIONS

1. A definite hazard incidental to the use of heavy weights on X-ray apparatus is shown to exist.
2. More attention should be paid by the manufacturers of X-ray apparatus to means of obviating such dangers.
3. Regular visual inspection can be relied upon to detect failure due to wear, but not to defective material.
4. It is advisable to have a periodical renewal of supporting cable.
5. Tube carriers should be locked when not in use.
6. One hand should firmly grasp the tube carrier when the latter is unlocked and over a patient.

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THE EFFECT OF IODIZED OILS ON SEROUS MEMBRANES[1]

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THE present study resulted from our attempt to make the heart more clearly visible under the fluoroscope by the injection of iodized oil into the pericardial sac. We were successful in obtaining very clear shadows by this method, but, to our surprise, the heart size seemed to increase from day to day and the animals died in the course of about two weeks. At autopsy, they showed pericarditis with marked effusion, although cultures of the pericardial fluid and smears from the heart surface were consistently negative for bacteria.

In view of the report of Sicard and Forestier (1), which concludes that Lipiodol is non-irritating upon injection into the epidural space, subarachnoid space, bronchial tree, male urethra, seminal vesicles, uterus, lacrimal ducts, and other regions, it was considered of some importance to follow up this evidence of marked irritative action upon the serous surface of the pericardium. Sicard and Forestier appear to consider their preparation quite innocuous, although they do not report intrapleural or intrapericardial injections; they do state that injection into joint cavities is productive of pain. Stolzner (2) and Pineas (3) each report one case where pathological changes followed upon the subarachnoid injection of iodized oil. Jarcho (4) believes that the injection of Lipiodol into the uterus and tubes is quite harmless. Blance and Livesay (5), using an iodized oil made by themselves, state that subcutaneous and intraperitoneal injections of the oil into guinea pigs cause no discomfort, nor does this substance produce any reaction in the peritoneum. Frazier and Glaser (6) find no ill effects in dogs from the injection of their own preparation of iodized rape-seed oil into the cerebral ventricles. In a personal communication Dr. B. H. Orndoff has told us that he observed a pleuritis in dogs following intrapleural injections of Lipiodol. A large proportion of the studies on iodized oils is concerned with visualization of the bronchial tree; this is an essentially different problem from that with which we are concerned and we have not attempted to review this literature.

METHOD

We have injected iodized oils into the pericardial sac, intrapleurally, intraperitoneally, and into joint cavities. Dogs have been used throughout. For intrapericardial injection the chest was opened under ether anesthesia, using artificial respiration and aseptic technic. The pericardium was grasped with two blunt hemostats and lifted out toward the incision, and 15 to 20 c.c. of iodized oil was injected into the sac, care being taken not to nick the heart surface with the needle. Upon removal of the needle a ligature which had been placed about the hemostats was immediately tied to prevent leakage. The incision was closed without leaving a pneumothorax. Intrapleural and intraperitoneal injections and injections into joints were made in the usual manner, with aseptic precautions.

A total of 10 intrapericardial injections of iodized oil was made in dogs weighing from 15 to 20 kilos. Eight of these were injections of 16 to 20 c.c. of Lipiodol (Lafay). Two were injections of 15 c.c. of Lipoiodin ("Ciba") in sesame oil. Lipiodol leaked through the ligature in two cases, and the pericardium ruptured a few days after

[1]Read before the Radiological Society of North America at the Fourteenth Annual Meeting, at Chicago, Dec. 3–7, 1928.

Fig. 1. Anteroposterior view of 5.7 kilo dog two days after receiving intrapleural injection of 15 c.c. Lipiodol.

Fig. 2. Anteroposterior view of 5.2 kilo dog 24 hours after intrapericardial injection of 20 c.c. Lipiodol.

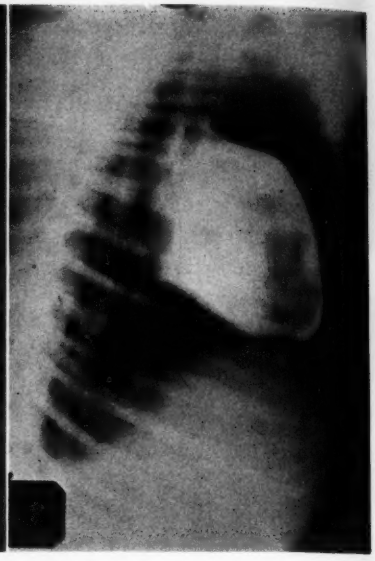

Fig. 3. Lateral view of 16 kilo dog 24 hours after intrapericardial injection of 18 c.c. Lipiodol.

one injection of Lipoiodin; in these cases, the oil was in contact both with the pericardium and the pleura.

Lipiodol was injected intrapleurally into five dogs varying in weight from 4 to 8 kilos, using from 16 to 20 c.c. for each injection. Two intrapleural injections of Lipoiodin in 10 c.c. amounts were made, using Lipoiodin in sesame oil for the first dog and in olive oil for the second. Intraperitoneal injections of from 17 to 20 c.c. of Lipiodol were made in four animals weighing from 5 to 10 kilos. Three dogs were used for joint injections, the right knee and shoulder joints of each animal being injected with 2 c.c. of Lipiodol.

As will be shown later, the pericardium was more susceptible than any other tissue studied to the irritant action of the iodized oils, hence all control injections were made intrapericardially. Lipiodol is iodized poppy-seed oil, while Lipoiodin is ethyl diiodobrassidate in solution in either sesame oil or olive oil. All of these oils were injected in the unhalogenated state as controls. Four intrapericardial injections were made of poppy-seed oil. For two of these injections we used the ordinary pharmaceutical product, and for the other two a purified poppy-seed oil of very low free fatty acid content which was prepared for us by the Abbott Laboratories. Similarly, we made four control injections of sesame oil, two of a relatively crude preparation and two of the purified product. Three injections of ethyl brassidate (prepared by the Abbott Laboratories) were made, and two injections of ordinary commercial olive oil.

Each animal was carefully autopsied after death, or was killed after a suitable time and an autopsy performed. Cultures and direct smears of the injected region were made in most cases, and, in several instances, frozen sections were made and stained with Sudan

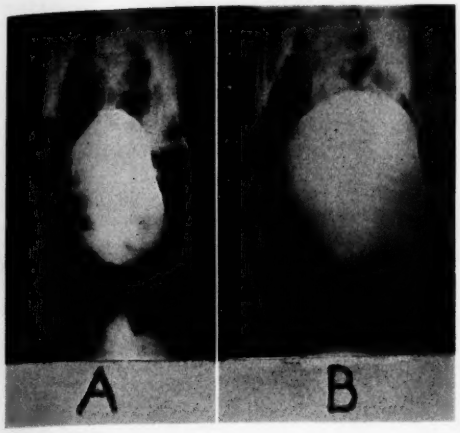

Fig. 4. Anteroposterior view of (*a*) 17.4 kilo dog two days after intrapericardial injection of 16 c.c. Lipiodol; (*b*) same dog seven days after injection.

III in addition to the usual hematoxylin and eosin preparations.

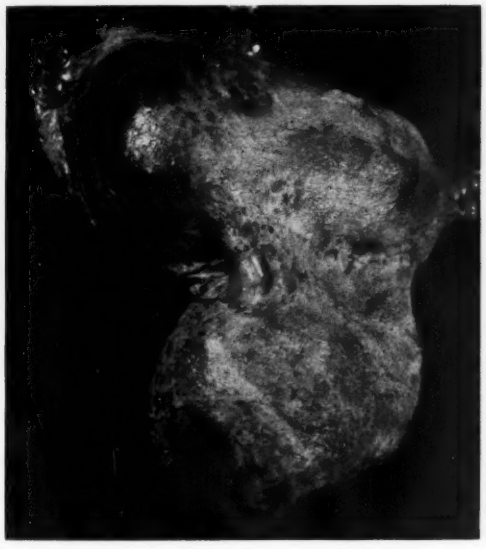

Fig. 5. Heart and parietal pericardium from dog dying nine days after intrapericardial injection of 18 c.c. Lipiodol.

RESULTS

Dogs which had received injections of Lipiodol or Lipoiodin into the pericardium died in from 5 to 16 days. If we discard those cases where the oil had leaked out into the pleural cavity, and one case where the animal contracted a post-operative pneumonia, the shortest length of life was 11 days and the longest 16. Two or three days before death the animals refused food, became very listless, and lost weight rapidly. Death was preceded by coma. At autopsy the animals showed definite thickening and inflammation of the pericardium, usually with a few adhesions on its external surface; the lungs were congested and edematous, or, in some cases, showed the picture of a pneumonia in the stage of red hepatization. There was commonly some fluid in the pleural cavity and the pleura was slightly inflamed. On opening the pericardium, from 90 to 300 c.c. of fluid was obtained. This fluid was creamy, consisting of oil emulsified in the serous exudate, and was occasionally blood-tinged. Both visceral and parietal surfaces of the pericardium were covered with a closely adherent mat of fibrin, and there were in most cases a few adhesions between heart and pericardium, especially in the region of the auricles. The whole picture resembled that of the "shaggy heart" of pneumococcus pericarditis. Cultures of the pericardial effusion and direct smears from the heart surface were made in seven cases, culture media used being glucose broth, agar and Loeffler's serum slants, and gelatin plates, but in no case did any growth occur nor did the direct smears show any bacteria. Direct smears of the effusion fluid always contained globules of oil, fibrin, and leukocytes. Frozen sections of parietal and visceral pericardia stained for fat with Sudan III, and, counterstained with hematoxylin and eosin, showed a heavy deposit of fibrin on the surface of the pericardium, and in the meshes of the fibrin were oil globules of all sizes. Directly beneath the pericardium we saw a few layers of heart muscle in which fatty degeneration had apparently occurred. Oil droplets which appeared be-

tween the muscle fibers were artifacts, having been carried in when the section was prepared. The sections stained in the usual manner with hematoxylin and eosin showed great numbers of leukocytes, chiefly lymphocytes, in the fibrin which covers the pericardium. Lymphocytes, together with a few red blood cells, were found in the pericardium and the tissue beneath it. There was no detectable difference between the findings in the Lipiodol-injected dogs and the animals where Lipoiodin was used.

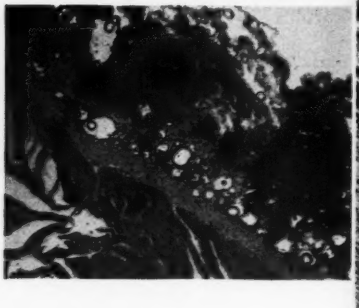

Fig. 6. Microphotograph of frozen section of visceral pericardium, stained with Sudan III. Note heavy fibrin deposit. Separation of muscle fibers is an artifact due to freezing. Dog died eleventh day after intrapericardial injection of 18 c.c. Lipiodol. (Fat photographs black.)

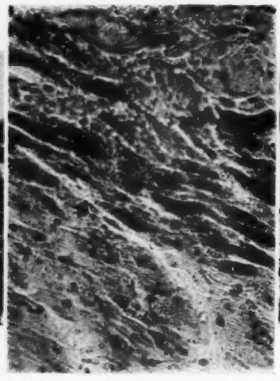

Fig. 7. High power microphotograph of same section as Figure 6. Note fat in muscle fibers directly beneath pericardium (stained with Sudan III). (Fat photographs black.)

Fig. 8. Microphotograph of frozen section of parietal pleura, stained with Sudan III. Fatty degeneration visible in muscle fibers directly beneath pericardium. Note heavy fibrin deposit containing many fat globules (fat photographs black).

The four dogs which received control injections of poppy-seed oil showed changes similar to those found in the animals which had received iodized oils; both the crude and the purified oils appear to be fully as irritating as iodized poppy-seed oil (Lipiodol). Again, in the animals receiving sesame oil intrapericardially we found no difference between the crude and purified specimens. The condition resulting from the injection of sesame oil, however, was unlike that produced by the other substances thus far studied in that the amount of fibrin formed on the pericardial surfaces was distinctly less; this we interpreted as signifying somewhat less irritant action. A pericarditis also followed the injections of ethyl brassidate in the three animals studied; in these cases the fibrin deposit was slightly less abundant and was not as friable. The two animals receiving olive oil showed no pathology except a small deposit of fibrin the size of a dime which formed over the site of the ligature on the parietal pericardium.

It should be noted that in several animals we observed the presence of ascites, while two dogs developed anasarca, with marked edema of all four extremities. The "nutmeg liver," typical of passive congestion, was not an uncommon finding.

Four of the animals which had received intrapleural injections of iodized oil died in from 8 to 12 days; the remaining three were killed 15 days after injection. All of the animals manifested loss of appetite, and listlessness. The autopsy findings in these animals showed much more individual variation than was present in those which had received intrapericardial injections. The ani-

mals which died spontaneously had more or less bronchopneumonia in addition to pleural findings. Those which survived to 15 days and were killed had the least pathology, namely, a slight inflammation of the pleural surface on the injected side, possibly a small amount of fluid exudate mixed with oil (which always remained), and a few adhesions of the lungs to the parietal pleura, the diaphragm, or the pericardium. Other animals showed a marked pleuritis with a fibrinous exudate over considerable areas of parietal and visceral pleura, as much as 80 c.c. of fluid in the pleural cavity, and many firm adhesions, especially about the base. Those animals in which intrapericardial injections had oozed out through the ligature gave the same picture as the intrapleurally injected dogs. Cultures and direct smears were made in four of these cases; no bacteria were found, while the effusion was seen to contain oil, fibrin, and leukocytes. Microscopic examination of tissue from the chest wall again showed a fibrin deposit on the parietal pleura which contained oil droplets, and also a fatty degeneration of a few muscle fibers just under the pleura. Leukocytes, chiefly lymphocytes, were present in the exudate, and in and about the pleura.

Wherever injections were made into the pericardium or pleura a marked enlargement of the regional lymph nodes, chiefly the mediastinal, occurred. This was especially noticeable in the dogs which had received intrapericardial injections; in these animals it was not uncommon to find masses of lymphoid tissue as big as a walnut in the mediastinum.

Of the four dogs injected intraperitoneally, one died of intercurrent pneumonia in 3 days; the Lipiodol was still in the abdomen and no pathology was present. The remaining three were killed in from 12 to 22 days. Autopsy showed a white oily fluid covering the peritoneum; there was no sign of inflammation, and direct smears from the peritoneal surfaces showed only oil with an occasional cell.

The three animals whose joints had been injected showed no symptoms. One was killed 22 and the others 26 days after injection. All showed substantially the same picture: the joints contained 1 to 2 c.c. of oily fluid, there was no change in the joint cartilage but microscopically there appeared to be a certain amount of fibrin deposit on the capsule. On microscopic examination, we found a membrane containing oil droplets on the capsule which was similar to that on the pericardium and pleura, although less extensive. The fluid in the joint was made up of oil, a mucin-like material, a small amount of fibrin, and leukocytes. All joints were sterile.

DISCUSSION

It is clear that while the iodized oils may be relatively harmless when in contact with many tissues of the body, this certainly is not true of the pericardium and pleura, and perhaps of the joint cavities. In the abdomen of the dog it appears to be without irritating action. As judged by the density of the X-ray shadow, it is so far absorbed from the peritoneum within three weeks that further irritant action would not be anticipated.

Poppy-seed oil and ethyl brassidate appear to be quite as irritating to the pericardium as their respective halogen compounds. We have no evidence that halogenation of oils increases their pathogenicity. Sesame oil is somewhat less irritating than the other substances studied, while olive oil appears to be almost without irritant effect on the pericardium. Control injections were not made elsewhere than into the pericardium because it appeared definitely established that this tissue is the most sensitive.

The iodized oils which we have used call forth the greatest reaction from the pericar-

dial tissue, less from the pleura, very little from the joint capsule, and none from the peritoneum. It is interesting to note that the pericardium is perhaps most actively in motion, the pleura less, the joints still less, and the peritoneal surfaces relatively little. This suggests that the amount of motion of a given tissue may be a factor in its susceptibility to injury by the iodized oils.

Since the pericardium appears to be one of the most sensitive tissues of the body to the action of oils, both plain and iodized, we suggest that intrapericardial injections of these materials be used as a method for determining the irritative action of recently developed preparations.

As by-products of this investigation, we present a method for the production of a sterile pericarditis with effusion, and a method for temporarily increasing the density of the heart shadow under the X-rays.

We wish to express our appreciation to Dr. A. Weil for his assistance in the preparation of microscopic material, and to the Abbott Laboratories for supplying us with various oils.

CONCLUSIONS

1. Two iodized oils—Lipiodol (Lafay) and Lipoiodin ("Ciba")—are irritating to certain membranes, as follows:

(*a*) Intrapericardial injection produces pericarditis with effusion, followed by death in about two weeks.

(*b*) Intrapleural injection produces a pleuritis of variable grade; some animals die.

(*c*) Injection into joints produces some inflammation of the capsule.

(*d*) Intraperitoneal injection appears harmless.

2. Poppy-seed oil and ethyl brassidate are equally as irritating to the pericardium as their iodine compounds; sesame oil is somewhat less irritating, while olive oil appears to be practically innocuous.

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GENERALIZED OSTEITIS FIBROSA[1]

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THERE are a number of bone diseases of unknown origin which, though presenting a varying clinical course, are nevertheless not only characterized by more or less similar deformities (due to softening, bending, and enlargement), but also show, in common, the fundamental histological picture of osteitis fibrosa.

There are many questions in reference to these diseases which clamor for an answer. On what basis can their relationship be established? Are they definite, isolated entities, characterized by a constant, clinical, roentgen, and microscopic picture, or are they but the varying aspects of one disease? Are there other bone dystrophies which can be shown as pathologically related to the generalized osteitis fibrosa group? What is the relationship of the mono-osteitic lesions of osteitis fibrosa to the generalized diseases? Are the accepted ideas regarding etiology, incidence, and genesis correct?

It is thus apparent that a correlation and a grouping of the clinical, pathologic, and roentgenologic facts bearing on generalized osteitis fibrosa are necessary in order to establish the significance of the polymorphous manifestations of this disease.

There has been much confusion in diagnosis, arising out of the desire to create entities not warranted by a broad pathologic outlook, and based on the prominence which one or another of the varied lesions of the disease may assume.

Thus leontiasis ossea[2] is often described as a separate and isolated disease, when in fact it is but osteitis fibrosa affecting the bones of the head and face. So also the chronic hyperplasias involving the superior maxilla or frontal bones producing massive, hard, bone tumor formations are but instances of mono-osteitic lesions of osteitis fibrosa. It would be as illogical to create separate entities for the varied pathologic manifestations of generalized osteitis fibrosa as it would be to consider gumma and the cirrhosis of syphilis separate diseases. Just as syphilitic periostitis may exist side by side with gumma of the bone, so in generalized osteitis fibrosa, the cyst and the giant-cell tumor may be found in juxtaposition and associated with simple fibro-osteoid changes.

It would appear, therefore, that much can be gained from a simplified classification based on a common pathologic relationship.

The X-ray examination is of paramount importance in the study of bone lesions in general and this group in particular, because it permits complete scrutiny of the entire bony system. Only in this way can an idea be obtained of the full distribution of the disease and of its development and genesis. The early stages can be found only in bones which are outwardly not deformed. Such lesions are overlooked in a necessarily limited pathologic examination of the fresh or macerated specimens.

In 1876, Paget (387) described a chronic generalized disease of bones occurring in persons past middle age. Though this was by no means the first description of this entity, since Wilks (567) in 1869 described a case under the title of osteoporosis, or spongy hypertrophy, and Czerny (104) in

[1]Read before the Radiological Society of North America, at the Thirteenth Annual Meeting, at New Orleans, Nov. 28–Dec. 2, 1927.

[2]Virchow did not give any histologic data bearing on this disease. He simply suggested this name as a collective one for diffuse hyperostosis of the skull. The assumption that an osteitis fibrosa was the cause of the change in leontiasis was first expressed by M. B. Schmidt (465). Prince (420) and Fitz (154) also held such a connection to be highly probable. Bockenheimer (61) maintains the identity of this disease with fibrous osteitis, based on microscopic studies of a typical case. M. Koch (268), von Recklinghausen (425), Boit (62), Telford (524), Lotsch (325), Langenskiold (301), and Durck (129) are also of this opinion. Jefferson (241), Parry (392), Myers (366), and Hamburger and Nachlas (210) have reported cases bearing on this relationship.

1874 described a lesion of the lower part of the tibia and fibula characterized by softening and bending, still Paget's description is so classical that his name is connected with the disease he termed "osteitis deformans."

In 1891, von Recklinghausen (425) described a chronic bone disease, osteitis fibrosa, with multiple tumors and cysts, occurring usually in young adults, accompanied by deformity and complicated by frequent spontaneous fractures. The tumors and cysts were considered as being end-stages of the osteitis fibrosa. Von Recklinghausen's name is thus brought into association with osteitis fibrosa.

In 1904, von Mikulicz (351) described a solitary cystic bone disease, involving the epiphyseal ends of the long bones. He believed this lesion to be an entity, characteristic of early childhood. He observed its benign character, with no recurrence or metastases, and noted the possibility of healing by consolidation after spontaneous fracture. The pre-cystic stage was one of a localized fibro-osteoid tissue change, with numerous giant-celled areas.

In 1910, von Recklinghausen (426) published an extensive pathological study in which he classified osteitis deformans and osteitis fibrosa (with and without cysts and tumors) with rickets and osteomalacia under the general heading of malacia.

The work of von Recklinghausen (426) on the various types of malacias and their significance is of fundamental importance in the roentgenologic analysis of the problems under consideration. This classification, by bringing the various bone dystrophies into relationship with one other, goes far toward making order out of the chaos. In spite of the fact that on minor points the conclusions of von Recklinghausen have been disputed, still his monumental work is, to one concerned with these bone dystrophies, a source of light and inspiration and tremendous help (Table I).

TABLE I

MALACIAS (VON RECKLINGHAUSEN)

(*A*) Porotic.
Porotic hypoplastic.
(*B*) Hyperplastic.
(*C*) Phlegmatoplastic (myelotic form of *B*).
(*D*) Metaplastic (Osteitis fibrosa, von Recklinghausen).
Bone cysts (non-parasitic).
(*E*) Hyperostotic metaplastic (Paget).
(1) Hyperostotic, porotic.
(2) Hyperostotic and cystic metaplastic, tumor-forming osteitis fibrosa.
(3) Benign sarcomas of bone (giant-cell tumors, epulis); neoplastic formation arising from osteitis fibrosa.
(*F*) Myeloplastic and hypostotic (osteogenesis imperfecta).

He designated rickets and osteomalacia, together with osteitis fibrosa and osteogenesis imperfecta, as belonging to one group—malacias, thus calling attention to the most striking physical characteristic of all the four diseases—softening of bone.

The great value of this coördinated grouping is that it permits the classification of all types of rickets, achondroplasia, osteoporosis and other types of generalized bone dystrophies, and gives, if nothing else, a working hypothesis as to their essential pathologic significance.

At first thought, it would appear that a histologic distinction between rickets, osteomalacia, and osteitis fibrosa (the three chief malacic diseases) could be easily drawn, but the more intensively and closely these lesions are studied the more difficult does it become to point out such sharp differences between them.[3]

In the anatomic conception, malacia (softening), and in the histologic picture, the metamorphosis of a part or the whole of one or several bones into fibrous tissue, the distinction which can be traced is a relative, not a specific one.

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[3]The characteristic of osteitis fibrosa is a fibrous issue change in the medullary cavity, replacing the medullary content and producing absorption of the cortex from within outward. In osteitis fibrosa the activity of the resorptive process and the fibro-osteoid substitution is marked, and there is the formation of mutiple tumor-like areas and cysts.

In osteomalacia decalcification is gradual. The osteoporosis advances from the center to the periphery; from the interior of the cancellous tissue, from the spongy substance surrounding the medullary canal or from the diploe,

Von Recklinghausen thus includes both the disease Paget described and the disease he himself described in the group of metaplastic malacias ("metaplastic" conveying the idea that the underlying process involves a complete reconstruction, the conversion of one tissue into another of similar morphologic origin and character), but calls Paget's form a *hyperostotic metaplastic malacia,* and osteitis fibrosa a *simple metaplastic malacia.* The metaplastic malacia includes all bone cysts except the parasitic variety.

Dawson and Struthers (112) have attempted to simplify this classification by calling von Recklinghausen's disease a simple metaplastic malacia, and the Paget's form a hyperplastic metaplastic malacia. Besides this, tumor-forming metaplastic and cyst-forming metaplastic are considered separate entities under the heading of metaplastic malacia. The benign sarcomata (epulis, myeloid, giant-cell tumors) are classified under metaplastic newgrowths. There are numerous transition stages between all these lesions.

In Table II the original classification is adhered to, with minor changes, and the pathologic phases of the various diseases are placed in schematic form.

The pathologic processes in Paget's disease and von Recklinghausen's disease are fundamentally similar, especially in the earlier stages. The conditions have in common a disturbance of the equilibrium which regulates the relation between the normal bone resorption and bone apposition.[4] This and the marrow changes finally lead to malacia. It would appear that the characteristic picture of the particular disease depends on the nature and intensity of the reconstructive effort. The constant change is the softening produced by resorption and fibro-osteoid tissue substitution, with resulting deformity. This osteoid tissue in Paget's disease becomes calcified and the new bone formed in old and new trabeculæ and under the periosteum produces the hyperplastic, hyperostotic changes, with sclerosis and eburnation. In von Recklinghausen's type, the newly formed tissue partly degenerates, forming cysts, and recalcification and ossification is minimal.[5] (Fig. 1.)

The "osteitis deformans" of Paget is, according to the usual description, a disease of late adult life; it runs a chronic course with bending and thickening of the bones; spontaneous fracture is rare; there is but slight tendency to the formation of tumor-

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toward the cortex. This is followed by a reversion to a fibrocellular connective tissue. Cystic areas form in rare instances. The new formation remains soft during the disease.

The striking general pathologic changes of rickets are imperfect ossification, excessive resorption of inner layers of the bone, fibrous tissue transformation of bone marrow, osteoid tissue formation—in part by decalcification and in part by metaplasia of cartilage and fibrous tissue.

[4]It might be well to briefly summarize the modern viewpoint of fundamental principles involved in the pathologic changes in bone. These ideas are most clearly expressed by Leriche and Policard.

The formation of bone is the result of a metaplastic change in the connective fundamental substance, and takes place in three stages: (*a*) transformation with a multiplication of connective fibrils; (*b*) infiltration by a special substance, chemically undefined—the pre-osseous substance; (*c*) deposits in that substance of a calcareous mixture of calcium phosphates and carbonates. This osseous transformation of connective tissue is an interstitial and humoral process independent of all cellular action. The cells present in the connective tissue prevent the formation of bone, by the product of their vital activity. Osseous metaplasia of connective tissue is a reversible process. Bone appears and disappears with the greatest facility. It is always in a state of unfixed equilibrium. As a result of rarefaction of bone, there is produced a localized oversupply of calcium which provokes an osseous metaplasia in the surrounding connective tissue. In the constructed bone, the bone cells have a minimum of vitality, and almost no metabolism. But, if their physiologic activity is resumed again, there is regression of bone (osteolysis) around them. The periosteum does not cover an osteogenic layer with numerous osteoblasts. When the periosteum is modified by a change of circulation or by edema, it then becomes a ground for ossification. It is passively ossified—it does not make bone in an active manner. Bone resorption takes place by osteolysis or osteoclasia.

In osteolysis, the osseous metaplastic transformation of the fundamental connective substance ceases and the connective matrix reappears. In osteoclasia, the action of phagocytic giant cells, the osteoclasts, occurs. They are unspecific, banal phagocytes, proceeding from evolution of osteoblasts. In the resorption of bone the osteoclasia plays a less prominent part than osteolysis. The resorption of bone is specially directed by humoral phenomena, dependent on the circulatory activity in the bone. The increase of activity of the circulation involves bone resorption.

[5]Virchow has established the fact that cyst formation in bone is never the primary occurrence, but that it is to be regarded as the result of the transformation of a primary alteration in bone structure.

Christeller maintains with von Recklinghausen that cysts and giant-cell tumors are the result of degenerative changes in the metaplastic and hyperostotic osteitis fibrosa. Originally there is a proliferating destructive fibrosis of the bone marrow which displaces the normal fat or hematopoietic fat. The proliferation may produce tumor-like fibrous masses. This new formation is often followed by regressive changes. The modes of origin of small cysts are related to degeneration (myxomatous softening), to productive proliferation (retraction of the fibrous tissue) and to hemorrhage and giant-cell formation. Trauma, which is put forth so prominently as an etiologic factor, may sometimes be indirectly responsible by injuring the bone marrow (marrow

like areas and cysts. Von Recklinghausen's disease, on the other hand, is considered to be a disease of early life, running a self-limited course. There is the formation of cysts and tumor-like areas and spontaneous fracture is common. No sharp and clear clinical or pathologic distinction can, however, always be drawn between them.

There are mono-osteitic lesions which closely resemble the changes of Paget's disease, and there are isolated lesions in the bones which closely resemble the changes of von Recklinghausen's disease. The long bones may show the changes characteristic of von Recklinghausen's disease, while the skull bones may have the typical appearance characteristic of Paget's disease.

There are localized lesions involving only a part of one bone, which show the changes of a simple metaplastic malacia (simple osteitis fibrosa), or of a cystic metaplastic malacia, or of a tumor-forming metaplastic malacia, or of a hyperostotic metaplastic malacia, or the same bone may show a combination of these typical changes.

The pathologic basis for the radiographic appearance lies in the following lesions:

(1) Fibro-osteoid changes;
(2) Malacia;
(3) Cyst formation;
(4) Giant-cell tumor formation;
(5) Hyperostosis.

These changes may exist as isolated lesions or they may exist in any combination.

Any of the lesions may involve one bone or more than one bone.

Any of the lesions may involve the entire bone or part of a bone.

The five changes above described can generally be determined roentgenologically.

1. The fibroid changes produce deviations from the normal in the medulla, cortex, and cancellated tissue. The periosteum is not involved. The medullary changes are shown by an increase in size and a change in form of the medullary area. The medullary area shows, more or less, expansion—either localized or involving the greater part of the bone. The fibulæ in Figure 3 show this clearly.

The changes in the cancellous bone are shown by a deviation in the architecture and density of the lamellæ, an increase in the size of the intralamellar spaces, and fusion of these spaces to form irregular areas of osteolysis. These are shown as illuminated, round, oval, or irregular areas free from bone markings, indefinitely outlined, with or without dividing septa. The changes in the cortex are shown by atrophy and lamellation. In later stages the cortical layer is reduced to a mere line of lamellar bone, covered by periosteum. The bone structure in the porous parts is honeycombed and there is extensive bone atrophy (decalcification).

2. The malacia is shown by softening, bending, deformity, and fracture. Any or all the bones may show the deformity resulting from the malacia. Even the arms and forearms are not uncommonly bowed. Fractures occur after the slightest traumatism. These spontaneous fractures are not accompanied by marked ecchymosis. The swelling is slight and the deformity varies. They usually heal without difficulty.

3. The term "cyst" is utilized to designate a certain uni- or multilocular cavity or hollow space in the bone. These symmetrical or asymmetrical cavities may be single or multiple. They are generally round or

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bleeding), the specific reaction product of which may be a localized osteitis fibrosa and cyst development.

Pommer, on the contrary, explains the production of cysts on a more or less mechanical basis. Bleeding within rigid bony walls prevents drainage of blood, embarrasses the circulation, and produces venous stasis, serous exudation and edema. These lead to increased pressure at the site and a pressure atrophy by lacunar resorption of the contiguous bone. This results in an extension of the space, with cyst formation.

Bone cysts may, in a general way, be divided into two groups:

(1) Those which bear a definite relationship to osteitis fibrosa. They may be unilocular or multilocular, with varying proportions of actual cavity and fibro-osteoid tissue.

(2) Those which are due to softening of giant-cell sarcomas, enchondromas, myxomas, callus and subperiosteal hemorrhage (scurvy).

The juxtaposition of areas of osteitis fibrosa, cysts, and giant-cell tumors has suggested to some the possibility that the disease runs through different stages and that the end-product is a cyst.

TABLE II.—MALACIAS

(1) Porotic *Rickets*[6] (usual type). *Osteomalacia* (Included in this group because fibro-osteoid reconstruction is minimal, but should really go under Metaplastic Group because the term "metaplasia" implies that the bone at one time possessed normal structure.)	Cortex: thinned Medulla: widened Trabeculæ: resorption through halisteresis
(*a*) Hypoplastic *Scurvy* (infantile) Moeller-Barlow type.	Exaggeration of porosity (bone resorption) Diaphysis: shortness and thinness Epiphysis: porosity related to deficiency in development of ossification centers. The postural deformities may occur in this group (coxa vara and valga, genu valgum and varum).
(2) Hyperplastic *Rickets* (variety of), with periosteal and medullary bone formation. *Chondrodystrophy foetalis.* Growth in length deficient, due to perversion of normal endochondral ossification.	Excess of bone formation Cortex and diaphyseal ends: thickened through osteoid tissue Medulla: narrowed by osteoid tissue Periosteal: osteophytes
(*a*) Plegmatoplastic Limitation of immature bone to the metaphyseal bone marrow. *Osteopetrosis.* (A diffuse malformation of bone and cartilage showing as a disturbance of ossification.)	Over-production limited to bone elements of endochondral origin Periosteal formation: slight Medullary cavity: network of osteoid tissue limited to metaphyseal bone marrow There is an increase of the cortex, with condensation and broadening of the trabeculæ, obliteration of spongiosa by the encroaching compacta, and obliteration of the marrow elements, with replacement by fibrous tissue. The periosteum is undisturbed. The hyperplastic bone is entirely endosteal.

All degrees of porotic, hypoplastic, and hyperplastic malacia may be present in the various forms of rickets

(3) Metaplastic. Complete reconstruction of bone, at one time normal

- (*a*) Simple Metaplastic *(osteitis fibrosa)*
- (*b*) Hyperostotic Metaplastic *(osteitis deformans, Paget)*
- (*c*) Tumor-forming Metaplastic Malacia (tumor-like areas in osteitis fibrosa)
- (*d*) Cyst-forming Metaplastic Malacia (cyst formations, solitary and multiple)
- (*e*) Metaplastic Newgrowths (benign sarcoma of bone, giant-cell tumor, epulis)[7] (not an essential part of osteitis fibrosa)

(4) Myeloplastic *Osteogenesis imperfecta.* Fetal and embryonal disturbance of first development of body tissues.	Over-production of marrow (hyperplasia) produces hypoplasia of bone Deficient endosteal and periosteal bone formation Bone reticular and porous Bone atrophy. Hypoplasia. Aplasia. Dysplasia.

[6] As is well known, there is a constant reconstruction of bone under normal conditions, resulting from evenly balanced bone apposition and bone resorption. In rickets there are not only the above-mentioned factors, but a gradual destruction of bone, a process called "thrypsis" by von Recklinghausen. If thrypsis prevails over apposition, a porotic type of malacia results; if apposition prevails, a hyperplastic type results. In rickets all degrees of and transitions between these two types may be present. The differences between rickets, late rickets, and osteomalacia are quantitative and related to the age of the patient, and show all transitions. The conception of the process underlying all is the same. The only essential difference between rickets and osteomalacia is the existence of endochondral disturbance in rickets and its absence in osteomalacia. In rickets the bones of the extremities are usually involved, while in osteomalacia it is chiefly the pelvis and vertebrae.

[7] Single benign tumors originating in the medulla, usually at the diaphyseal end of long bones, and tumors arising from the periosteum (giant-cell epulis). These tumors are circumscribed, not markedly infiltrative, highly vascular and friable; resemble granulation tissue, and show cystic degeneration. According to von Recklinghausen they are not tumors but metaplastic new formations originating in a fibro-osteoid tissue, thus relating these lesions to localized osteitis fibrosa. This is in contradiction to Barrie's view that these are chronic inflammatory processes, chronic hemorrhagic osteomyelitis, and fibrocystic osteomyelitis.

oval in shape, cortical or medullary in location, and vary greatly in size. They are definitely localized, sharply circumscribed, have more or less of a definite covering, and may contain fluid or granules. Cysts may become organized and completely ossified.

Roentgenologically, the cystic areas cannot always be differentiated from those occupied by cartilaginous rests.

4. The tumor-like areas are usually intimately associated with cysts. The tumors originate in the cortex and protrude into the medullary cavity as they enlarge, but always their cortical origin may be easily recog-

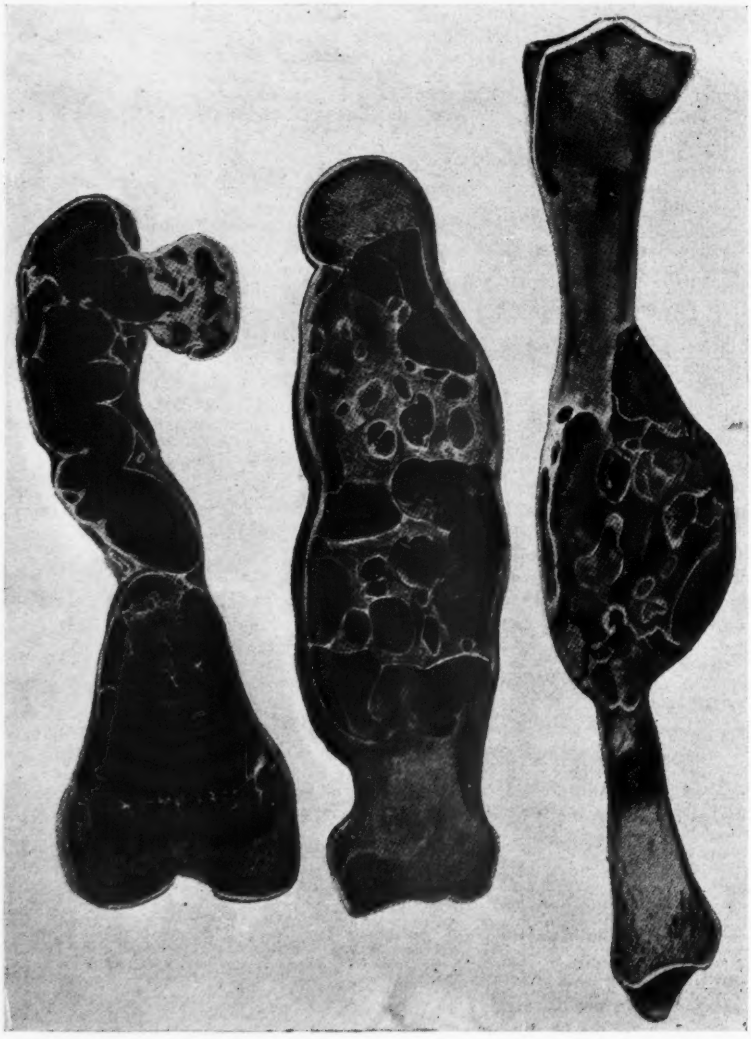
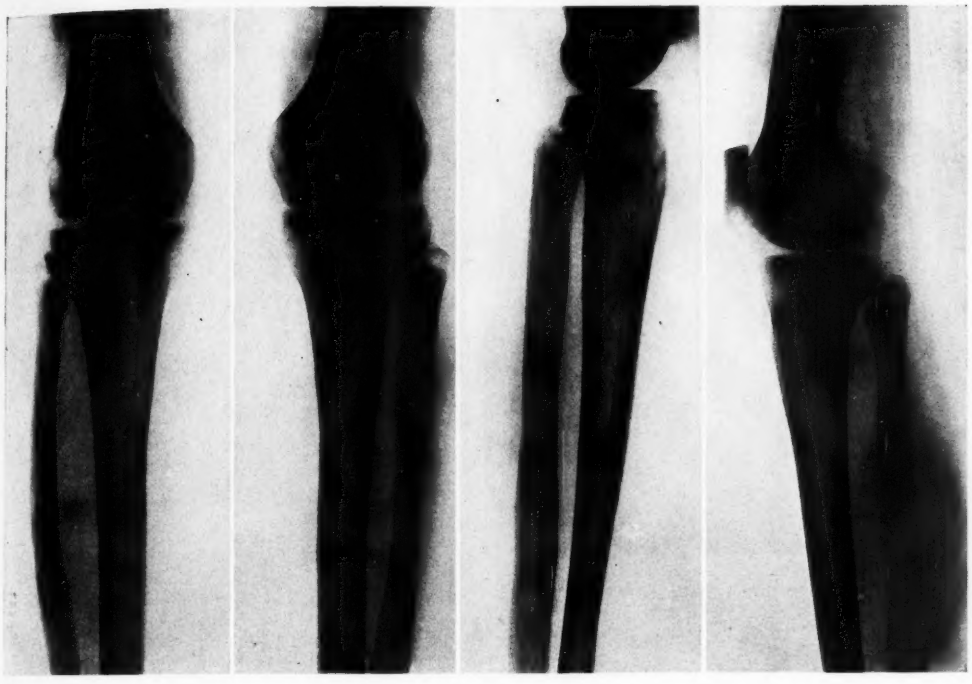


Fig. 1. Metaplastic cyst- and tumor-forming malacia, showing the changes in the femur, humerus, and tibia (Stenholm). The basis for the radiographic appearance is here clearly apparent. (See page 507.)



Figs. 2 and 3. Diffuse metaplastic changes of the fibula, with bowing. Note the irregular thickening of the tibia.

nized. They produce no periosteal or cartilaginous destruction and are always sharply localized, sharply limited, and do not infiltrate into soft tissues, in spite of considerable expansion. They may originate in any portion of the bone. They may be centric or excentric in their relationship to the shaft. The contour of the tumor masses is not sharp when bordering on areas of simple fibro-osteoid changes, but clear and definite where ossification of the fibrous tissue has taken place. The full extent of the tumor involvement is frequently obscured by hyperostotic changes.

The roentgen differentiation between a giant-cell tumor and a true sarcoma is frequently impossible. At certain stages the morphologic characteristics are exactly the same.

5. When the ossification takes place in the medulla, it is in the form of long longitudinal bands which appear to be endosteal and merge with the cortex, though of definitely less density. The cancellated structure shows the productive bony changes both as irregular bars or bony septa and as spherical nodules of dense bone.

The cortex is thickened by the formation of irregularly placed, generally longitudinal bars of newly formed bone, the distinction from the medulla being sharply contrasted. Where the fibro-osteoid changes have been extensive and the ossification not complete, all distinction between compacta and medulla is lost. The entire bone has the appearance of a rough weave of bone lines, with wide meshes. Sometimes the meshes are small and the bone lines close together and denser in the cortical area. Thus, the homogeneous smooth-textured compacta, which as a result of atrophy and fibro-osteoid tissue growth has become fibrillated, is

Fig. 4. Localized simple metaplastic malacia of the lower end of the radius. Note the sharp demarcation of the involved area.

Fig. 5. Mono-osteitic form of simple metaplastic malacia of radius.

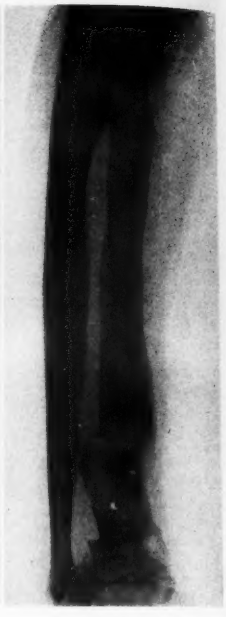


Fig. 6. Diffuse metaplastic malacia of the radius, with fracture of the lower end. The lesion involves the entire shaft and extends to the epiphysis. There is a beginning cystic degeneration.

gradually transformed by osteoplasia into a coarsely reticulated structure, dotted with irregular dense patches.

When ossification of the cystic areas takes place, the illuminated central zone may become completely obliterated and replaced by a ball of dense bone, in which, after a time, the regular lamellar structure may be seen. The bordering wall of the cystic area is, however, nearly always to be made out.

The ossification of a tumor area results in the obliteration of the loculations and a combined new bone formation from within and without the mass.

The disease appears to go through the stages of fibrosis, degeneration, cyst formation, and re-ossification. It would appear that the progress of the lesion is slow; that it develops to a certain stage and then either re-ossification takes place or it remains for years quiescent and unchanged, showing no further encroachment on the normal bone. Elsewhere, however, new lesions may appear and go through the various stages to healing.

The changes in the bones of the skull and of the hands and feet deserve special consideration. With all varieties of generalized osteitis fibrosa, the skull may show no changes or a simple asymmetry. On the other hand, the vault bones may be normal while the bones of the base may be involved symmetrically or asymmetrically. Involvement of a single face bone is not unusual. The boundary line between involved and normal parts may be defined by an elevated ridge of bone. However, the skull may show the following changes:

(1) *Simple metaplasia:* Diffuse decalcification over a large area, with marked thinning of the tables (Fig. 7), a variety of craniotabes, like senile osteoporosis.

(2) *Hyperostosis:* A generalized, diffuse, homogeneous thickening of both tables

Fig. 7. Characteristic skull changes of the simple metaplastic form of generalized osteitis fibrosa. There is a marked loss of density of the lateral portion of the skull, the area being definitely delimited. This lesion might be characterized as a "craniotabes."

Fig. 8. Characteristic changes of the hyperostotic form of metaplastic malacia (Paget's disease). The increase in the thickness of the bones of the vault, the porosity, and the new bone formation in the form of irregular plates and spherical masses are apparent from the frontal eminences to the occipital protuberance. Note the elevation of the spheno-basilar area, and the deepened and narrowed posterior fossa. In reality, there is a total lowering of the base of the skull on the spine, which accounts for the attitude.

Fig. 9. Localized hyperostotic changes in the frontal bone.

and base of the skull, with a diminution in the size of the diploëtic layer, or its bony obliteration (Fig. 4). This may be localized to the frontal, parietal, or occipital areas.

(3) *A combination of metaplastic and hyperostotic changes:* This is the type best known. There is marked widening of the diploëtic layer, which is filled with irregularly distributed bony masses. The outer table may be thinned and the inner very much thickened, or both may be thickened (Fig. 8). This process may be general or localized.

On the other hand, there may be no discernible changes in the skull and yet nearly every bone in the body may be invaded by the disease (Figs. 10, 11, 12).

The radiographic appearance of the skull is generally pathognomonic. Usually, this is also the most striking outward change. The marked asymmetry, general enlargement, and the deformity are highly characteristic. The fronto-occipital circumference may attain a dimension of 70 cm., and the thickness of the cranial vault may be as much as 7.5 cm. In spite of the large volume, the weight of the skull is remarkably low, due to the loose, spongy structure of the bony masses. The bone is very brittle. There is a striking contrast between the gigantic skull and the atrophic, senile facial development.

The inner and outer tables become porous, with numerous minute perforations, which give the bone a worm-eaten appearance in contrast to the smooth surface of the normal skull. This roughening is increased over the internal surface by the existence of small, irregular osteophytic projections and by the deepened linear depressions for the meningeal vessels.

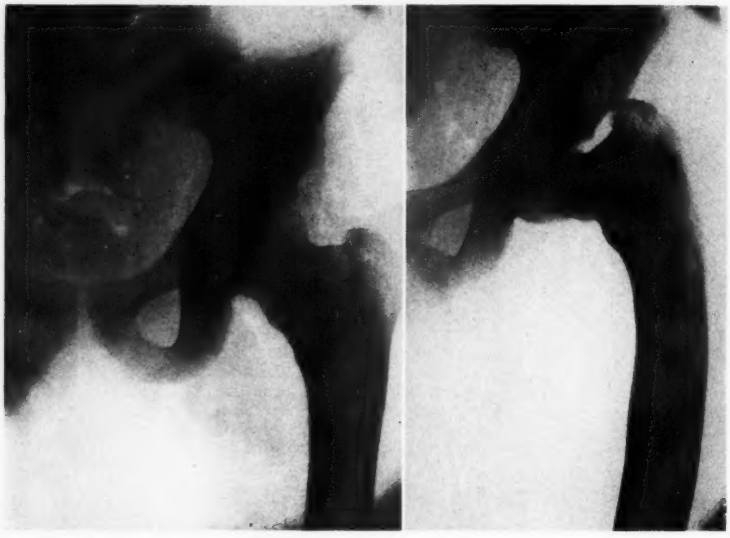
The changes in the basal portion of the skull are common and striking. As in the long bones, they are the results of the combined malacia and hyperostosis. There

may be an enormous thickening of the base, particularly of the sphenoid and the basilar portion of the occipital bone, and, as a result of the malacia, a depression of the anterior and posterior fossæ about the spheno-basilary kyphosis. The sphenoidal sinus may be obliterated by bone thickening and the sella turcica may become extremely shallow and flattened. The petrosa and mastoid portions of the temporal partake in the thickening. The new bone production results in a narrowing of all the foramina.

The frequency with which the bones of the hands and feet are involved in generalized osteitis fibrosa is not appreciated. There is scarcely a case in which some sign of the disease cannot be found in these bones, and in doubtful cases the lesions in these bones may give the clue to the disease. The involvement may be fairly symmetrical. The first three metacarpals are most commonly involved, especially the second and third.

The lesions are usually small or minute, but, nevertheless, distinctive. There may be merely a coalescence of the lamellæ at the base of one of the metacarpals, with the formation of an enlarged interlamellar space, or this may be definitely circumscribed by a demarking ring of bone, constituting a cyst formation.

There may be extensive osteoporosis of the phalanges, with thinning of the cortex, or there may be clear-cut defects in the cortex. There may be irregular thickenings of the cortex of isolated areas or of isolated bones.



Figs. 10 and 11. Generalized metaplastic cyst-forming malacia. Note the complete absence of changes in the skull (Fig. 12), the cystic changes involving the femora on both sides, coxa vara of both femoral necks, and the involvement of the ilium. (J. T. Murphy.)

Part II

Paget considered the disease he described as one of middle life. Packard, Steele, and Kirkbride (385), in a study of 51 cases, found the average age to be forty-nine and one-half years. The youngest in their series was a patient of Moizard and Bourges (355), in whom the disease seemed, in their

opinion, to have quite definitely started at the age of twenty-one. The age of onset, as given in the majority of cases reported, is usually in the fourth or fifth decade. Stilling (509) reported three cases in which the patients were 70, 77, and 92 years old, respectively.

Instances of involvement at early age have, however, been reported: Sonnenberg (497), 16 years; Elsner (137), 12, 13, 14, and 16 years; Thibierge (526), 13 years; Hartmann (217), 14 or 15 years; Jones (244), 16 years; Klestadt (262), a patient first seen at 35 whose head had been large from birth; Vogel (550), a patient seen at 68 years whose head had always been large and ill-formed; Da Costa's (106) case, in which the head involvement was quite definitely dated as beginning at 8 years.

A survey of the literature makes it evident that well-marked examples of the disease have been found from childhood to extreme old age, and that in a number the markings of the disease, as judged by gross deformities, are apparent at birth.

Further, if the histories of the reported cases are studied, it will be found that the given period of onset is based on the phenomena of pain or disability and gross deformity. It is obvious that the disease is a slow, insidiously progressive bony change, generally symptomless in its development; that it must take a definite number of years to develop, and that before the patient's attention is called to it the most striking changes have already taken place. The error of judging the period of onset by the subjective manifestations or on the basis of deformity is therefore apparent.

It is obvious that if a patient at 48 gives the history that the trouble began twelve years before with a bowing of the leg, the disease cannot be said to have had its onset at 36; or when a patient first appears complaining of pain in the legs for a period of one year and at that time the X-ray examination shows a full-blown bony involvement with all its classical features, the duration of the disease cannot be said to have been one year. It takes more than one year —more than twenty years—for the average case to develop to the stage of marked deformity. Besides this, there are periods when the disease seems to be in abeyance. In Moizard and Bourges' (355) patient the disease progressed slowly for fifty-two years before death. It is a common experience to diagnose Paget's disease by general external conformation and find extensive bony involvement in patients who have had no symptoms whatever referable to the bony system, or to note a certain bone deformity which shows the characteristic changes, and to be told that the deformity had existed "always" but no attention had been paid to it.

Fig. 12. See caption under Figures 10 and 11.

In order to arrive, therefore, at a correct idea on this point it is important to know how long in the average case it takes for the disease to develop its definite bony changes. These studies can be made only roentgenologically. No correct deductions can be made from subjective and objective criteria. It would be necessary to have a

Fig. 13. Simple cystic multilocular metaplastic malacia of the upper end of the humerus, fractures

(Conclusion of legend)

at the upper and lower ends of the involved area. In these cases the multiple cystic area may extend to the joint surface and protrude into it. The multiple form occurring as it does in malacic, porotic bone may perforate the cortex and even the joint surface. When healing takes place the involved areas are filled in by masses of bone. The fracture lines may show either as light lines, or, when the fragments are impacted, as dense lines. The roentgenographic appearance of trabeculation is often produced by irregularities in the walls surrounding the lesion.

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roentgenologic record of the incipient changes and to study the case until the disease is in its fully developed stage. Such records have not been consistently made. Though the first published radiographic study is by Watson (554) (1898), who reported the seventh case in America, there are numerous cases reported thereafter in which no mention is made of the X-ray examination.

Where no such radiographic studies have been made we are confronted by the difficulty of determining whether these cases were of the hyperplastic or the metaplastic type. It is not possible to judge the variety of the bony changes from the outward conformation. The attitude and general deformity are strikingly similar.

That the metaplastic type has as one of the characteristics of its clinical picture definite manifestation early in life, is well appreciated.

(To be concluded in July)

Editor's Note:—Owing to the extensiveness of this paper it has been divided into two sections. The reference figures following the names refer to the Bibliography, which will be found at the end of the paper in the July issue.

EDITORIAL

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FOUNDATIONS IN ROENTGENOLOGY

In many lines roentgenology stands supreme as a method of diagnosis, and yet the very features which make it supreme have not been stressed to the point that the average roentgenologist has recognized them. In the differentiation of the benign from the malignant lesion, roentgenology presents the evidence in indisputable form in the great majority of cases, and yet one of the most frequent errors of the average practitioner is the failure to read the evidence as it is presented.

The roentgenologic sign of malignancy, whether it is in bone or in the gastro-intestinal tract, is a break in the contour of the normal shadow and a sharp demarcation of the normal from the abnormal. One has but to study the pathologic specimen after tissues have been removed from the body and correlate the pathologic picture with that of the roentgenogram to get the explanation of this. The ulcerating surface seen in carcinomatous gastro-intestinal tissues corresponds exactly with the irregular, serrated edge seen in the film, and the transition from the pathologic to the healthy tissue is macroscopically visible.

In bone one has to deal with secondary (metastatic) carcinoma, with primary malignant involvement, usually sarcoma, less frequently with the Ewing type of tumor and with myeloma. The remarkable thing, again, is the sharp demarcation of the normal from the abnormal that is seen in the cortex. Myeloma constitutes one exception to this statement. In secondary carcinoma two distinct forms are recognized: the osteoclastic, or purely destructive type, and the osteoplastic, in which, added to destruction, there is tissue proliferation with increased density of the bone as seen in the roentgenogram.

No more vivid description of the osteoclastic type than "melted ice" has been offered. If the bony structure can be visualized as formed of ice, the lesion resembles that produced by the direction of a ray of heat by a blowpipe against the involved part. The osteoplastic type presents a coarsely granular shadow showing coincident destruction and proliferation. This can be distinguished from the homogeneous density seen in osteitis sclerotica (marble bone) and the sequelæ of typhoid and syphilitic infections sometimes seen in the pelvic bones. Clinically marked secondary anemia associated with abnormal density of the bones suggests marble bone.

In nearly all benign lesions there is a tendency to enlargement of the cortical shadows; in malignant lesions there is never apparent or real enlargement of the contours of bones.

In sarcoma there is usually elevation of the periosteum at the line of demarcation, denuding of the periosteum below this, and non-encapsulated "invasion" of the soft tissues coinciding with the denuded area. In Ewing's type of tumor the periosteum frequently is laminated, giving the "onion skin" appearance, and when there is invasion of the soft tissues the outlines of the tumor just suggest encapsulation: they are not well

defined as in the benign lesion, neither are they completely invisible as in sarcoma.

In myeloma the pathognomonic characteristic is multiple punctate areas of destruction, with smooth margins, just as if they had been punched out with a metal instrument. In the flat bones and in the humerus, one may see expansion of the contour, the bone retaining all the characteristics of a benign tumor.

In all benign lesions of bone or of the gastro-intestinal tract the contours of the roentgen shadow remain intact. The margins of any defect are smooth throughout and never indistinct, serrated, or irregular, as they are in malignant lesions. This may serve to differentiate secondary (metastatic) malignant lesions and fibrosarcomas which do not involve the cortex. In the gastro-intestinal tract the papillary types of carcinoma must always be kept in mind as exceptions to the rule; because of lack of ulceration these may retain the roentgenologic characteristics of benign tumors.

When the roentgenologist assumes the responsibility of deciding the malignancy or non-malignancy of a given lesion, he should be guided entirely by the roentgenographic data. Anteroposterior and lateral views are always necessary in long bones. Stereoscopic films of pelvic bones will be found more useful. Palpatory manipulation, with the patient at various angles, is an imperative added factor in examination of the gastro-intestinal tract.

Correlation of the roentgenographic with the clinical data should follow the roentgenographic examination. In cases of doubt the use of the tourniquet and excision of a specimen for microscopic study should always precede a decision to amputate. Any other procedure may be disastrous to the interests of the patient and most embarrassing to the roentgenologist.

Frequent experiences of seeing benign tumors in which amputation has been advised and urged would lead one to believe that there remains much to be done in the education of our profession in interpretation. A careful study of the films with emphasis on the points mentioned, followed by correlation of the roentgenographic with the clinical data, will do much to strengthen the opinion of the roentgenologist in this field of diagnosis.

CHARLES G. SUTHERLAND, M.B. (TOR.)

RESOLUTION PERTAINING TO EXCLUSIVE LEASES

ADOPTED BY THE CHICAGO ROENTGEN SOCIETY

WHEREAS, there are at this time several large buildings in the "Loop" and adjoining business district of Chicago which have negotiated exclusive leases for space in which to practise roentgenology, and

WHEREAS, if the aforementioned practice of giving exclusive leases in this district be continued and extended to buildings now in the course of construction and being planned, there will be practically no desirable space available for roentgenologists, and

WHEREAS, the specialty of roentgenology is a medical branch and a true medical specialty, as much so as is surgery, neurology, ophthalmology, gynecology, pediatrics, orthopedics, etc., and is consequently of paramount importance to public and private health, in the diagnosis and treatment of disease, and

WHEREAS, exclusive leases are never even considered, much less asked for, by any of the other medical specialties, or given to any of them, and

WHEREAS, this giving of exclusive leases is discriminatory against roentgenologists,

because of the reasons previously herein set forth, therefore, be it

RESOLVED, by the Chicago Roentgen Society, that the giving of exclusive leases in any building, to any medical specialists, is wrong, is in bad form, is an unjust discrimination against that specialty, and, it is further

RESOLVED, that the Building Managers Association of Chicago be and are hereby most earnestly and urgently requested to cease the issuance of any exclusive leases to any medical specialist, be he surgeon, psychiatrist, gynecologist, pediatrician, or roentgenologist, and that at the termination of existing exclusive leases these be not renewed, and it is further

RESOLVED, that copies of these resolutions be transmitted to the aforementioned Building Managers Association of Chicago, the Chicago Association of Commerce, and to the editors of RADIOLOGY, the *American Journal of Roentgenology and Radium Therapy*, the *Bulletin of the Chicago Medical Society*, and to the Council on Medical Education and Hospitals of the American Medical Association, the American College of Surgeons, the American College of Radiology, and the American College of Physicians.

ATTEST:

WALTER T. BRONSON, M.D.
Secretary-Treasurer.

SECTION ON RADIOLOGY

The Scientific Assembly of the Section on Radiology of the American Medical Association will meet in Portland, Oregon, July 10, 11, and 12, one-half day session on each day.

Radiologists who are anticipating attendance upon this annual Assembly may look forward to a program of varied interest, to be presented by men of outstanding ability and wide experience in the subjects presented. The program will be printed in the *Journal of the American Medical Association* for June 8, 1929, and in the July issue of RADIOLOGY, out in June.

X-RAY AND RADIUM PROTECTION[1]

RECOMMENDATIONS OF INTERNATIONAL CONGRESS OF RADIOLOGY

Abstract.—The X-ray and radium protection proposals that were adopted by the Second International Congress of Radiology are given in their final form. The section of the 1928 National Electrical Code, dealing with X-ray and high frequency apparatus, is also given. The first set of recommendations are effective until the next congress in 1931.

The Bureau receives many requests for information regarding safety and protection from X-rays and radio-active substances. In answering these, each case has been considered in itself and, when possible, recommendations have been made. At the Second International Congress of Radiology in Stockholm, 1928, there was adopted the following series of recommendations (not fixed rules) covering such protection, and it is intended that from time to time additions or modifications will be made conforming to the advances in the practice and applications of radiology. In connection with these recommendations it should be understood that in the various protection coefficients the minimum value consistent with safety have been given.

The general form of the proposals is that adopted several years ago by the British X-ray and Radium Protection Committee and

[1]This is Circular of the Bureau of Standards No. 374, reprinted by permission of Mr. George K. Burgess, Director.

which in the meantime has been found applicable and quite satisfactory.

I. RECOMMENDATIONS ADOPTED BY THE SECOND INTERNATIONAL CONGRESS OF RADIOLOGY, JULY, 1928

1. The dangers of overexposure to X-rays and radium can be avoided by the provision of adequate protection and suitable working conditions. It is the duty of those in charge of X-ray and radium departments to insure such conditions for their personnel. The known effects to be guarded against are: (*a*) Injuries to the superficial tissues; (*b*) derangements of internal organs and changes in the blood.

1. Working Hours, Etc.

2. The following working hours, etc., are recommended for whole-time X-ray and radium workers:

(*a*) Not more than seven working hours a day.

(*b*) Not more than five working days a week. The off-days to be spent as much as possible out of doors.

(*c*) Not less than one month's holiday a year.

(*d*) Whole-time workers in hospital X-ray and radium departments should not be called upon for other hospital service.

2. General X-ray Recommendations

3. X-ray departments should not be situated below ground-floor level.

4. All rooms, including dark rooms, should be provided with windows affording good natural lighting and ready facilities for admitting sunshine and fresh air whenever possible.

5. All rooms should be provided with adequate exhaust ventilation capable of renewing the air of the room not less than ten times an hour. Air inlets and outlets should be arranged to afford crosswise ventilation of the room.

6. All rooms should preferably be decorated in light colors.

7. X-ray rooms should be large enough to permit a convenient layout of the equipment. A minimum floor area of 250 square feet (25 square meters) is recommended for X-ray rooms and 100 square feet (10 square meters) for dark rooms. Ceilings should be not less than 11 feet (3.5 meters) high.

8. A working temperature of about 18° C. (65° F.) is desirable in X-ray rooms.

9. Wherever practicable the X-ray generating apparatus should be placed in a separate room from the X-ray tube.

3. X-ray Protective Recommendations

10. An X-ray operator should on no account expose himself unnecessarily to a direct beam of X-rays.

11. An operator should place himself as remote as practicable from the X-ray tube. It should not be possible for a well-rested eye of normal acuity to detect in the dark appreciable fluorescence of a screen placed in the permanent position of the operator.

12. The X-ray tube should be surrounded as completely as possible with protective material of adequate lead equivalent.

13. The following lead equivalents are recommended as adequate:

X-rays generated by peak voltages	Minimum equivalent thickness of lead
Not exceeding—	mm.
75 K.V.	1.0
100 K.V.	1.5
125 K.V.	2.0
150 K.V.	2.5
175 K.V.	3.0
200 K.V.	4.0
225 K.V.	5.0

14. In the case of diagnostic work, the operator should be afforded protection from scattered rays by a screen of a minimum lead equivalent of 2 mm.

15. In the case of X-ray treatment the operator is best stationed completely outside the X-ray room behind a protective wall of a minimum lead equivalent of 2 mm. This figure should be correspondingly increased if the protective value of the X-ray tube inclosure falls short of the values given in Paragraph 13. In such event the remaining walls, floor, and ceiling may also be required to provide supplementary protection for adjacent occupants to an extent depending on the circumstances.

16. Screening examinations should be conducted as rapidly as possible with minimum intensities and apertures.

17. The lead glass of fluorescent screens should have the protective values recommended in Paragraph 13.

18. In the case of screening stands the fluorescent screen should, if necessary, be provided with a protective "surround," so that adequate protection against direct radiation is afforded for all positions of the screen and diaphragm.

19. Screening stands and couches should provide adequate arrangements for protecting the operator against scattered radiation from the patient.

20. Inspection windows in screens and walls should have protective lead values equivalent to that of the surrounding screen or wall.

21. Efficient safeguards should be adopted to avoid the omission of a metal filter in all X-ray work.

22. Protective gloves, which should be suitably lined with fabric or other material, should have a protective value not less than ½ mm. lead throughout both back and front (including fingers and wrist). Protective aprons should have a minimum lead value of ½ mm.

4. *Electrical Precautions in X-ray Rooms*

23. The floor covering of the X-ray room should be insulating material, such as wood, rubber, or linoleum.

24. Permanent overhead conductors should be not less than 9 feet (3 m.) from the floor. They should consist of stout metal tubing or other coronaless type of conductor. The associated connecting leads should be of coronaless wire kept taut by suitable rheophores.

25. Wherever possible earthed guards should be provided to shield the more adjacent parts of the high tension system. Unless there are reasons to the contrary, metal parts of the apparatus should be efficiently grounded.

26. The use of quick-acting double pole circuit breakers is recommended. Overpowered fuses should not be used. If more than one apparatus is operated from a common generator, suitable overhead multiway switches should be provided.

27. Some suitable form of kilovoltmeter should be provided to afford a measure of the voltage operating the X-ray tube.

5. *Radium Protective Recommendations*

(A) Radium Salts

28. Protection for radium workers is required from the effects of:

(*a*) Beta rays upon the hands;

(*b*) Gamma rays upon the internal organs, vascular and reproductive systems.

29. In order to protect the hands from beta rays, reliance should be placed, in the first place, on distance. The radium should be manipulated with long-handled forceps, preferably made of wood, and should be carried from place to place in long-handled boxes, lined on all sides with about 1 cm. of lead. All manipulations should be carried out as rapidly as possible.

30. Radium, when not in use, should be stored in a safe as distant as possible from the personnel. It is recommended that radium tubes or applicators be inserted into

separate lead blocks in the safe, giving a thickness of protective wall amounting to 5 cm. of lead per 100 mg. of radium element.

31. A separate room should be provided for the "make-up" of screened tubes and applicators, and this room should be occupied only during such work.

32. In order to protect the body from the penetrating gamma rays during handling of the radium, a screen of not less than 1 inch thickness of lead should be used, and proximity to the radium should occur only during actual work, and for as short a time as possible.

33. The measurement room should be a separate room, and it should contain the radium only during its actual measurement.

34. Nurses and attendants should not remain in the same room as patients undergoing radium treatment.

35. All unskilled work or work which can be learned in a short period of time should preferably be carried out by temporary workers, who should be engaged on such work for periods not exceeding six months. This applies especially to nurses and those engaged in "making up" applicators.

36. Discretion should be exercised in transmitting radium salts by post. In the case of small quantities it is recommended that the container should be lined throughout with lead not less than 3 mm. thick. It is more satisfactory to transport large quantities by hand in a suitably designed carrying case.

(B) Emanation

37. In the manipulation of emanation, protection against the beta and gamma rays has likewise to be provided.

38. The handling of emanation should be carried out, as far as possible, during its relatively inactive state.

39. The escape of emanation should be very carefully guarded against, and the room in which it is prepared should be provided with an exhaust fan.

40. Where emanation is likely to come in direct contact with the fingers, thin rubber gloves should be worn to avoid contamination of the hands with active deposit. Otherwise, the protective measure recommended for radium salts should be carried out.

41. A separate pumping room should be provided with a connecting tube from the special room in which the radium is stored in solution. The radium in solution should be heavily screened to protect persons working in adjacent rooms. This is preferably done by placing the radium in solution in a lead-lined box, the thickness of lead recommended being according to the following table:

Quantity of radium element, in grams	Thickness of lead	
	inches	cm.
0.5	6.0	15.0
1.0	6.6	16.5
1.5	6.8	17.0
2.0	7.2	18.0

II. Discussion of the Recommendations

The proposals do not specify the use of sheet lead, but state that a certain "lead equivalent" be used. Sheet lead is, however, the safest and most permanent protection, and should be used whenever possible. If protective plasters are used, great care must be taken in their preparation and application in order to insure a uniform protective value. Papers by Hunt[1, 2] discuss the properties and use of such materials.

In most X-ray departments the lighting is inadequate and the ventilation poor. These are two factors which are very com-

1F. L. Hunt and M. Temin: Radiology, February, 1927.
2F. L. Hunt: Am. Jour. Roentgenol. and Rad. Ther., 1925, XIV, 524.

monly overlooked in the design of X-ray departments and consequently particular attention should be given to Paragraphs 4 and 5.

There is frequently also a lack of sufficient auxiliary protective aprons on screening stands and couches, thus exposing the operator to the very soft radiation scattered from the patient's body. This is one of the most common sources of danger to the doctor.

In the edition of 1928, the Electrical Committee of the National Fire Protection Association formulated Section 5012 of the National Electrical Code, which bears directly on protection from X-ray equipment. Some measures in this code are covered in the proposals above, but where there is any difference between them, the more stringent should be followed. This section follows.

III. 5012. X-RAY AND HIGH FREQUENCY APPARATUS

(*a*) Adequate mechanical barriers shall be provided to prevent too close approach to any high voltage part except the operating tube and its leads, and it is recommended that all other parts be inclosed in a separate room or cabinet. Such barriers may consist of grounded metal or of insulating material, such as glass. High voltage parts inclosed in a wooden cabinet shall have adequate spacing from the wooden walls. If one side of the high voltage circuit is grounded, the milliammeter shall be connected in the grounded lead, and need not be guarded. All operating parts, such as spark gap handles and regulating handles, shall be made of suitable insulating material, and shall be operative from outside of the barriers.

(*b*) Overhead high voltage stationary conductors shall be not less than 7 feet 6 inches above the floor where the ceiling height permits, and in no case less than 7 feet. The high voltage leads on tilting tables and fluoroscopes shall be adequately insulated or so surrounded by barriers that inadvertent contact with them is improbable. . Tube terminals and high voltage wires connected thereto should be adequately insulated for a distance of 12 inches from the terminal. Shields for this purpose shall be designed to carry the high voltage leads away from the patient in a direction at right angles to the long axis of the tube. X-ray tubes used in therapy shall be mounted in a grounded metal inclosure.

(*c*) The low voltage circuit of a step-up transformer shall contain a manually operable automatic circuit breaker having no exposed live parts. There shall be an additional switch in this circuit, which for diagnostic work shall be one of the following types: (1) A switch with spring or other mechanism to open automatically except while held closed by the operator; (2) A time switch which will automatically open after a definite time for which it has been set.

(*d*) Where more than one piece of apparatus is operated from the same high voltage source, each shall be provided with a high voltage switch so as to give independent control.

(*e*) Low frequency current-carrying parts of machines of the quenched-gap or open-gap type shall be insulated or guarded so that they can not be touched during operation. This applies to all parts except the high frequency circuit proper which delivers high frequency current normally for therapeutic purposes.

(*f*) Transformers which are a part of an X-ray or high frequency apparatus, even though they contain oil, are to be considered and treated as a part of the device, and need not conform to the requirements of Section 5005 for power transformers.

(*g*) All tube stands and fluoroscopes shall have their frames, operating handles, and other non-current-carrying metal parts of apparatus grounded in conformity with the requirements of Article 9. Non-metallic tables and chairs are recommended, particularly for therapeutic work. Metal or other conducting tables or chairs for supporting patients shall be suitably insulated from ground, and insulating floors, mats, or platforms shall be provided for operators. It is recommended that floors of concrete or other conducting material be completely covered with linoleum, rubber tile, or other insulating material.

IV. DISCUSSION

With the possible advent of new X-ray tubes, such as the "Metalix tube," and new technic, such as high tension cables, and oil-immersed equipment, many of the problems in protection would be simplified. However, the present type of equipment will undoubtedly be used for some time, and, therefore, proper attention should be given to all new installations as well as existing ones.

In order to continue the work in protection, the Congress has elected a committee of international character which will report any further progress at the Third International Congress of Radiology at Paris in 1931. This committee consists of one representative each from England, France, America, Germany, Sweden, and Italy, and to this committee will be referred any final decisions regarding protection. In this country it is planned to have a small "local" committee which will decide upon recommending any changes or additions arising from experience and advance in the United States. To this committee should be referred any questions or suggestions regarding X-ray protection and, if no settlement can be reached, they will be taken up by the international committee, of which Dr. G. W. C. Kaye and Dr. Stanley Melville are the honorary secretaries. Communications pertaining to the code should be transmitted through Lauriston S. Taylor, Bureau of Standards, the American member on this committee.

Washington, September 15, 1928.

BOOK REVIEWS

UROLOGICAL ROENTGENOLOGY. By HUGH H. YOUNG, M.D., Clinical Professor of Urology, Johns Hopkins University; Director, James Buchanan Brady Urological Institute, Johns Hopkins Hospital, and CHARLES A. WATERS, M.D., Instructor in Clinical Roentgenology, Johns Hopkins University; Assistant Visiting Roentgenologist, Johns Hopkins Hospital. Volume VII of Annals of Roentgenology, published by Paul B. Hoeber, Inc., New York, 1928. Pages 499. Price $20.00.

As the authors somewhat surprisingly state in the preface, "Urological Roentgenology" is not confined to the roentgenographic aspect of urology but "includes a brief but comprehensive outline of the whole subject of urology, with stress on the pathology and the gross and minute changes externally and internally which they produce, and also a brief outline of the symptomatology, diagnosis, and therapy." The inclusion of so much extraneous subject matter readily obtainable from standard text-books is to be regretted as it reduces markedly the room available for urological roentgenograms and renders the book less handy as a ready reference.

Thus, we find the first chapter given over to the history of the development of urological tables and instruments, while the second deals with the physiology and anatomy of the urinary tract, and so a considerable portion of the text is consumed before the subject matter in hand is commenced.

Aside from the redundancy this inclusion compels, the book contains a large assortment of roentgenograms that illustrate the greater number of conditions, both normal and pathologic, that are usually encountered in roentgenologic work in the urinary tract.

The space allotted to the various topics could have been better proportioned, as the

chapter on lithiasis occupies more space than is devoted to neoplastic disease, infections, infestations, and tuberculosis combined. The interpretation of roentgenograms dealing with these conditions usually requires more study than of those associated with lithiasis.

The inclusion of charts relating to urinary output, surgical technic, and the proper application of radium in the rectum when encountered seems out of place in a work on roentgenology, but as these charts tend to broaden the readers' conception of the subject they are not a needless addition. Most of the illustrations are accompanied by case histories which add to their interest, and the habit of accompanying many of the roentgenograms with an artist's drawing of the pathologic material following operation is to be especially commended.

The last chapter in the book, entitled "Neurological Lesions of the Urinary Tract," although of but sixteen pages, merits particular attention. The illustrations are excellent, the text is pertinent, and the subject well covered.

ROENTGENOLOGY: THE BORDERLANDS OF THE NORMAL AND EARLY PATHOLOGICAL IN THE SKIAGRAM. By ALBAN KOEHLER, Prof. Dr. Med., Wiesbaden, Ex-president of the German Roentgen Society, Honorary or Corresponding Member of the Swedish Medical Society, Vienna Medical Society, American Roentgen Ray Society, Spanish and Mexican Radiological Society, member of the British Institute of Radiology, Honorary President of the International Congress for Electrology and Radiology in Amsterdam, 1907, member of the Executive Committee of the Society of German Scientific and Medical Researches. Rendered into English from the fifth German edition by ARTHUR TURNBULL, M.A., B.Sc., M.B., CH.B. (Glasg.). New York, William Wood and Company, 1928. Price $14.00.

ROENTGENOLOGY: ITS EARLY HISTORY, SOME BASIC PHYSICAL PRINCIPLES AND PROTECTIVE MEASURES. By G. W. C. KAYE, O.B.E., M.A., D.Sc., F. Inst. P. With 49 illustrations. Paul B. Hoeber, Inc., New York. Price $2.00.

Two vastly different books under the same title engage our interest. Kaye's book is small and is really his Caldwell Lecture before the 1927 American Roentgen Ray Society at Montreal. Kaye is a leading physicist of England and is Director of the National Laboratory for Physical Experiments. He has just been made President of the British Roentgen Society.

Koehler's larger book is an exposition of normal anatomy as displayed by the roentgenographic method. This is the first English translation, although there have already been five German editions. Koehler has achieved international fame through this book and many other roentgen studies. He has been President of the German Roentgen Society.

Koehler's book is of great practical value to the roentgen world. Kaye's is historical and philosophical, but with certain practical points in X-ray protection.

Koehler's excellent volume takes up each bone and anatomical organ of the body, describes its usual normal roentgen appearance together with the normal variations of development, contour, and growth. It is really a roentgen anatomy. The book is prefaced with commendations by Dr. John Fraser, of Edinburgh, the surgeon; Dr. John Woodburn Morison, radiologist at the Royal Infirmary, and Dr. James T. Case, of Chicago. Dr. Arthur Turnbull, of Glasgow, has done this excellent translation. Without intent of plagiarism but with full agreement upon these expressions of praise, your reviewer must draw from their sentiments.

As Koehler prefaces, this book is for beginners (?) in roentgenology or the inex-

perienced. The work is an adviser on the diagnosis of those findings which exhibit or appear to exhibit slight and not particularly noticeable divergencies from the normal anatomical picture. For such cases there has been no complete work in the roentgen literature. The atlases of normal anatomy show only roentgenograms of the usual everyday typically normal conditions, while the atlases of pathological anatomy contain only pictures of very pronounced diseases, recognizable as such without any difficulty. Whether, for example, a small bony process, an indentation, a roughness, a small translucency or opacity of the spongiosa structure, a thickening the size of a pea in the shadow of certain soft tissues are to be reckoned as belonging to the normal or the pathologic, is a question no one ever asks. To recognize and correctly diagnose these slight alterations is quite as important as, indeed, more important than, the diagnosis of pronounced diseases; for the earlier a disease is diagnosed, the more successful can its treatment be. On the other hand, we are enabled to avoid regarding quite normal findings as pathologic ones; for example, to mistake the fabella in the external tendon of the gastrocnemius for an articular body may result in operations harmful to the patient.

From the American prefacer, Dr. James T. Case, we quote the following: "The English-speaking section of the world-fraternity of radiologists will join in gratitude to the author and to the translator for making this valuable dictionary and atlas of radiologic criteria available in our own language. We feel certain that the book will find a very hearty welcome. It is needed not only by specialists in radiologic diagnosis, but by general surgeons, orthopedists, urologists, anatomists, gastro-enterologists, specialists in pulmonary diseases, as well as by physicians and lawyers whose work brings them in contact with problems of legal medicine. There is no other work in any language which contains all the facts pertaining to radiologic interpretation so patiently and painstakingly collected, digested, and organized for ready and easy reference in this book."

It is easy to agree with, even amplify, the splendid encomium offered by the several prefacers. Dr. Koehler has produced a reference roentgeno-anatomy that will receive enthusiastic praise from all who use it. So many of its good points crowd upon one for recital that it seems useless to choose from among them within the limits of this review.

Kaye's small volume carries historical notes, well illustrated, upon the early philosophers, upon the nature of the roentgen rays, total reflection of roentgen rays, prismatic refraction, diffraction by ruled gratings, and the nature of radiation. Then follow three chapters upon X-ray protection. In his concluding chapters upon the future of roentgenology, he says: "The three phenomena of the differential absorption of roentgen rays by various kinds of matter, the biological effects of the rays, and the diffraction of rays by crystalline solids have already been turned to account in the arts, sciences, and industries in many directions. Specialization will become inevitable, as is apparent from the immense literature that has already come into being from the activities of the large body of workers who have been attracted by the various problems to which roentgen rays are being applied, whether medical, scientific, or technical. Our knowledge of the ultimate structure of the solid state of matter already owes more to roentgen-ray analysis than to any other means. As to the value of roentgenography to the surgeon, it is surely almost incalculable. On the biological side it would seem that, while wonderful results are achieved from time to time in therapy, progress is somewhat hampered by a number of factors."

The appendix carries the recommendation of the British X-ray and Radium Protection Committee, which was adopted *in toto* by the International Congress at Stockholm in July, 1928.

E. H. S.

ABSTRACTS OF CURRENT LITERATURE

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On the Roentgen Therapy of Priapismus in Leukemia. Paul Krause. Strahlentherapie, 1928, XXXI, 125.

According to the author, there are about fifty cases of priapismus in leukemia on record. He relates the detailed history of two patients, both suffering from myelogenous leukemia, who presented that symptom. X-ray therapy locally as well as to the spleen, thigh, and arms brought considerable relief. The pathology and the probable mechanism of the X-ray effect are also discussed.

E. A. POHLE, M.D., PH.D.

Massive (Atelectatic) Collapse of the Lung: With Report of Case Showing Associated Spontaneous Pneumothorax. L. R. Sante. Am. Jour. Roentgenol. and Rad. Ther., September, 1928, XX, 213.

A case of massive atelectatic collapse of the lung is reported which occurred during an asthmatic attack in a male patient, age 19, which at the time of the original X-ray study showed an unusual complication, a spontaneous pneumothorax. The pneumothorax probably occurred through the over-expansion and rupture of a small uncollapsed portion of the upper lobe. The treatment to relieve the distressing dyspnea and cyanosis consists in simply turning the patient onto the unaffected side and inducing him to cough. This usually releases the obstruction in the bronchus and allows a prompt re-inflation of the lung.

J. E. HABBE, M.D.

Clinic and Roentgenotherapy of Omarthritis. S. Grauer. Strahlentherapie, 1928, XXIX, 172.

Arthritis of the shoulder joint was treated by applying three surface doses of 7 H each (about 300 *R*) in seven-day intervals, through an anterior or posterior field of 20 × 20 centimeters. The minimum wave length amounted to about 0.1 Ångstrom, the filter to 0.63 millimeter Zn. + 2.0 Al. In most cases the effect was very encouraging, the inflammatory type particularly responding immediately and being relieved permanently.

E. A. POHLE, M.D.

Further Investigations Regarding the Changes of the Functional Stage of the Blood Vessels Following Roentgen Irradiation. N. W. Lazarew. Strahlentherapie, 1928, XXIX, 602.

Ears of rabbits were irradiated with doses leading to epilation or inflammatory reactions, sometimes followed by atrophy of the skin. After ten months, the animals were killed. Pharmacological tests of the blood vessels in the exposed areas showed a decrease in constriction following injection of histamin and caffein. This is in agreement with the observations on the human skin after exposure to roentgen rays.

E. A. POHLE, M.D., PH.D.

A Statistical Study of Clinical and Laboratory Findings in Gastric and Duodenal Ulcer, with Special Reference to Roentgenologic Data. T. Grier Miller, Eugene P. Pendergrass, and Katherine S. Andrews. Am. Jour. Med. Sci., January, 1929, CLXXVII, 15.

The authors reviewed and analyzed the data in 279 operatively proved gastric and duodenal ulcer cases, with a selected group of 36 gastric and 100 duodenal cases. The duodenal cases are four times as frequent as the gastric. The male sex predominated and the women were a little older than the men. The history was of the greatest diagnostic importance aside from the roentgenologic findings; the physical examination less so. Fifty per cent showed a localized spot of tenderness in the epigastrium. High gastric acidity was found in both types of cases, but also in normal individuals.

The roentgenologic findings were: (1) Diagnosis was correct in 94 per cent in gastric ulcer and 88 per cent in duodenal ulcer; (2) The most important sign was the filling defect; (3) In 23 cases diagnosed either pyloric stenosis, gastroduodenal ulcer, or pyloric obstruction, only 6 ulcers seemed to be at the pylorus—the rest were duodenal; (4) Healed ulcers give defects similar to adhesions or active ulcers, but do not present the associated fluoroscopic phenomena.

ROBERT A. ARENS, M.D.

The Value of Roentgen-ray Therapy in Primary Malignant Tumors and Benign Giant-cell Tumor of Bone. William A. Evans and T. Leucutia. Am. Jour. Roentgenol. and Rad. Ther., October, 1928, XX, 303.

The authors present a series of twelve cases, most of them registered by the Bone Sarcoma Registry, as primary malignant bone tumors or benign giant-cell tumors, in all of which cases deep roentgen irradiation played an important, or even the exclusive, part in effecting a two- to six-year cure of the patient. Summaries of the cases are as follows:

Case 1 (Registry No. 526). A highly malignant spindle-cell sarcoma of the ilium was resected twice and then given deep roentgen therapy. When the patient died two and one-half years later the microscopical findings were those of a slow-growing osteochondrosarcoma.

Case II (not classified by Registry). Clinically and roentgenologically an osteogenic sarcoma of the frontal and sphenoid bones, treated solely by deep irradiation. The patient was alive and clinically well five years later.

Case III (Registry No. 523). Clinically, roentgenologically, and histologically a cellular osteogenic sarcoma of the ilium. The patient was alive and well eight years after onset and six years after development of lung metastases.

Case IV (Registry No. 889). An osteogenic sarcoma of the upper end of the fibula.

The patient was alive and well five years after onset of symptoms. Treatment: Pre-operative irradiation, resection, post-operative irradiation for recurrence.

Case V (Registry No. 524). A tumor of the maxilla, roentgenologically malignant but histologically probably essentially inflammatory (Ewing). The patient was well five years after roentgen and radium irradiation.

Case VI (Registry No. 888). Ewing's tumor, with multiple bone lesions; first tumor noted four years previous to report. The patient was well one year after X-ray treatments.

Case VII (not classified by Registry). Tumor of hard palate variously diagnosed endothelioma, round-cell sarcoma, etc.; treated by radium, X-ray, and, later, curettement and post-operative irradiation. The patient was well three years after discontinuing treatment.

Case VIII (Registry No. 533). Multiple chondromata, with sarcomatous degeneration, treated by irradiation. Condition roentgenologically stationary and patient symptom-free four years after treatment.

Case IX (not registered). A myxoma of the metacarpal well five years after curettage and post-operative irradiation.

Case X (not classified by Registry). Patient operated on in 1924 for tumor of ilium. Frozen section showed spindle-cell sarcoma. Treated by post-operative irradiation. Two years later bone regeneration of ilium; patient symptom-free. (Patient died later from perforated appendix.)

Case XI (Registry No. 528). A giant-cell tumor of the lower end of the femur infected by biopsy. Failure of roentgen irradiation in presence of infection. Amputation necessary.

Case XII (Registry No. 529). Giant-cell tumor of lower end of femur. The patient was clinically well, with marked regeneration of bone, four years after completion of three series of X-ray treatments.

It is the authors' opinion that the cases reported constitute decisive evidence that roentgen therapy is one of the most powerful agents available for the treatment of malignant bone tumors.

J. E. Habbe, M.D.

Contribution to the Knowledge of the Roentgen Hyposensitivity of the Skin. Ludwig Haas. Strahlentherapie, 1928, XXIX, 608.

This is an interesting article because it relates the history of a girl who is less sensitive to roentgen rays than the average person. While cases of hypersensitivity or even idiosyncrasy to roentgen rays are well known, very few of lowered sensitivity have been published. The patient in question was suffering from severe hyperhidrosis of both hands. An erythema dose through 2 mm. of aluminum had no effect; at intervals of six to eight weeks repeated treatments were given, going as high as 265 per cent S.U.D. in one single sitting. Even at that high dose, no erythema followed; there was only slight pigmentation. Eight months after the last treatment there were no other changes.

E. A. Pohle, M.D., Ph.D.

The Diagnosis of Cancer of the Stomach. William Gordon. Brit. Med. Jour., Dec. 29, 1928, No. 3547, pp. 1163.

Practically all clinically discoverable gastric malignancies are primary, and almost all are carcinomatous. Gastric sarcoma is a rare disease, probably not found in more than 5 per cent of the cases. Gastric cancer is now the commonest of all cancers in England, and is most frequent between the ages of forty and sixty. The duration of symptoms varies widely, extending from three months to four years, the average being about one year. Symptoms which have lasted more than eighteen months, unless then associated with wasting and cachexia, are unlikely to be due to cancer, but there are exceptions. The author believes that the present observations indicate that the occurrence of cancer is unusual in gastric ulcers and it is an important clinical truth that most patients with cancer have not suffered previously from any serious gastric disorder.

Significant symptoms, which are of the utmost value, are: (1) indigestion, such as discomfort after meals, fullness, weight, nausea

and pyrosis; (2) marked anorexia, which is considered of great importance; (3) weakness, when it is in excess of that which might be caused by the anorexia; (4) cachexia; (5) anemia; (6) pain, which may be absent in 13 per cent of the cases and which is rather variable; (7) vomiting, which, like pain, is absent throughout the disease in about 13 per cent of the cases; (8) hemorrhage, which is shown by hematemesis in about 33 per cent of the cases, but which is practically constant in some degree and will be shown at least by occult blood in the stools; (9) the palpable tumor, which is found sooner or later in about 75 per cent of the cases; (10) fever, which occurs in about 33 per cent of the cases at some time or other; (11) offensiveness of breath and vomitus; (12) diarrhea; (13) dysphagia; (14) enlarged supraclavicular glands, which are quite rare; (15) edema of the feet, which is a late symptom; (16) thrombosis of the veins, regarding which some German writers have asserted that when in doubt about the diagnosis of cancer the appearance of thrombosis of veins anywhere makes cancer probable, and (17) "occult blood" in the stools, if constant, is most suggestive of cancer.

Analysis of the gastric content is often essential. Stress is laid on: (1) absence or marked diminution of free hydrochloric acid, but notation is made that free hydrochloric acid is absent in some cases of gastritis, in gastric atony, pernicious anemia, and in acne rosacea; (2) presence of lactic acid; (3) foul odor; (4) blood; (5) pus; (6) fragments of growth, and (7) the presence of Sarcinæ.

The X-ray findings are of the utmost value in many cases, and when available should invariably be used. The author refers to the fact that in the hands of unskilled roentgenologists they may be misleading.

The "cardiac sign," which was described by the author in 1903, has been confirmed by twenty-five years' experience. He says it is almost pathognomonic of carcinoma and that only two other conditions can produce it—starvation and diarrhea. This sign consists of a diminution, which may go on to complete disappearance, of the deep cardiac dullness in the recumbent posture, when there is nothing else to account for it. This sign is apt to be conspicuous in gastric carcinoma cases.

The sites of secondary growths vary. Fifty per cent occur in the liver, 35 per cent in the peritoneum and omentum, 19 per cent in the pancreas, and 10 per cent in the lungs and pleura. Therefore, sooner or later, jaundice is found in about 15 per cent of the cases, ascites in 25 per cent, and pleural effusion in about 10 per cent. Twenty-five per cent of liver cases are secondary to stomach cancer, and gastric cancer is perhaps the commonest cause of peritoneal malignancy. Other secondary growths, occasionally useful in diagnosis, are skin nodules and bone metastases, as revealed by the roentgen ray.

Certain complications must be borne in mind also in order to avoid mistakes. General peritonitis occurs in about 3 per cent, and has been recorded as the first indication of cancer. A gastric perforation into the general peritoneal cavity is a rare event. Perigastric abscess occurs in about 3 per cent and may be subphrenic, epigastric, hypochondriac, or umbilical. Gastric fistula is found in less than 3 per cent. Cerebral or spinal symptoms are found in 1 per cent of the cases and extension of the growth may, in rare cases, result in abscesses in liver, spleen, or kidney.

The diseases which simulate cancer and cause the commonest difficulties in diagnosis are chronic gastritis, gastric or duodenal ulcer or their sequels, hepatic cirrhosis and gastralgia.

Other tumors which may be confused with gastric cancer are: (1) cancer of the colon, which may lead to visible peristalsis and strongly suggest gastric cancer, but from which differentiation can be made by the X-ray; (2) cancer of the head of the pancreas may give many of the gastric symptoms, but numerous signs, such as fat in the stools, Cammidge's test, and Loewi's test, should differentiate; (3) pyloric cancer may simulate tumor of the omentum and X-rays should be the best guide here to a certain diagnosis; (4) simple gastric tumor, of which the differentiation must be made by exploratory incision; (5) the possibility of a hair ball must not be

forgotten in the case of an hysterical person; (6) feces in the colon, which, the author states, may closely simulate gastric tumor; (7) aneurysm, which may cause confusion—and such error is easier to make than one might imagine, and (8) the possibility of gumma must be borne in mind.

In conclusion, the author says that four points must never be forgotten, namely: (1) it is never justifiable to open the abdomen as the sole means of making a diagnosis; (2) it is never justifiable to refrain from advising laparotomy when there is reasonable hope of successful removal; (3) the diagnosis must be arrived at by a joint use of clinical, radiological, pathological, and, if necessary, surgical means (of these, the greatest are the clinical); (4) "dyspepsia" is not a diagnosis.

H. J. Ullmann, M.D.

On the Radiation Therapy of Pelvic Inflammatory Diseases. H. Kamniker and St. Simon. Strahlentherapie, 1928, XXX, 441.

This is a report of 81 cases of pelvic inflammatory disease, 30 acute, 17 sub-acute, and 34 chronic cases, which were treated by small doses of roentgen rays (150 K.V., 0.5 Zn. + 1.0 Al., 10 to 45 per cent surface dose or 2.5 to 13 per cent effective in the diseased tissue). The results were best in acute cases (70 per cent were improved or cured), while among the chronic cases only 35 per cent were improved. A dose of 10 per cent E.D. gave the highest percentage of results, while with a dose of 45 per cent E.D. they dropped to the lowest point. Amenorrhea following treatment and improvement of the condition in the adnexa did not always run parallel. Ambulatory treatment is not advisable. The percentage of recurrences is high in chronic cases, in those of gonorrheal origin, and following massive X-ray doses. Sometimes the condition of the patient is worse immediately after exposure but improvement is usually seen later—from several hours to two days later. The effect of roentgen therapy is somewhat similar to that of protein therapy.

E. A. Pohle, M.D., Ph.D.

Osteoma of the Frontal Sinus: Report of Two Cases, Robin Harris. The Laryngoscope, May, 1928, XXXVIII, 331.

After a brief review of 115 cases of true osteoma of the frontal sinuses previously reported, the author adds two more of his own, making a total of 117. He is of the opinion that osteoma of the frontal sinuses is the result of an inflammatory process due to chronic sinusitis, although trauma at times may be an etiologic factor. He then reviews "Attachment," "Growth and Size," "Diagnosis and Symptoms," X-ray examination being an invaluable aid in the diagnosis and exact determination of size and character of growth.

The two cases reported by Dr. Harris were admitted to the United States Veterans Bureau Hospital, No. 88, in April and September, 1926, respectively. In both cases the tumor was of such size that marked deformity was present, with displacement of the eye in both cases. The tumor was removed, with excellent results, the deformity being markedly improved. Recovery from the operation was rapid and uneventful in both cases.

B. C. Cushway, M.D.

Some Results of a Study of Roentgenograms of the Abdominal Viscera. Robert O. Moody and Roscoe G. Van Nuys. Am. Jour. Roentgenol. and Rad. Ther., October, 1928, XX, 348.

The writers made a study of the livers and spleens of 1,200 students of the University of California. Films were made with the patient standing at a target film distance of 90 cm., and with the patient prone and supine on the Bucky diaphragm at 72.5 cm. distance. The average liver length was 21.1 cm. in males and 20.3 cm. in females. Normal long livers may have their lower tip 5.0 cm. below the interiliac line. The excursion of the liver upon changing the patient from the vertical to the horizontal position varied from 0 to 10.9 cm., with the average excursion being slightly less than 3.0 cm.

The average length of the spleen was found to be 12.9 cm., with variations ranging from

7.0 to 12.0 cm. The width of the spleen was a somewhat less accurate measurement roentgenographically and was found to show variations from 3.5 cm. to 10.0 cm. in this series of measurements, with the average being 5.0 to 6.0 cm. In more than half the males and females the lower pole of the spleen was opposite the level of the third lumbar vertebra, which is fully two vertebræ lower than the position given in text-books on anatomy.

The livers may be long and the spleen short, or, *vice versa,* the liver may be short and the spleen long, and such variations may not be necessarily in direct relation to the size of the individual.

Thirteen subjects were put through a two-minute running exercise and in every instance the spleen shadow showed a recognizable decrease in size immediately following the effort.

Spleen shadows, likewise, were found to diminish in size immediately following the removal of blood for transfusion from two individuals.

J. E. Habbe, M.D.

The Advance of Diagnosis in Relation to Duodenal Ulcer. R. W. McQuay. Can. Med. Assn. Jour., May, 1928, XVIII, 543.

This contribution is reviewed because of the following statement made by the author: "A record of some twenty-six cases kept by me, in which the X-ray findings of one of the leading roentgenologists on the Continent were negative, showed that the clinical history was so conclusive that they were recommended for surgery. In twenty-two of these duodenal ulcer was demonstrated at operation."

In view of the generally accepted value of the X-ray in the diagnosis of duodenal ulcer, it would seem that the author makes an unwarranted aspersion upon his radiological associates. This conclusion seems confirmed by the author's statement farther on in the paper, that in 134 cases of duodenal ulcer, the X-ray findings were definitely positive in 120 cases.

L. J. Carter, M.D.

Experience with the X-ray Therapy of Hypertrophy of the Prostate. Rudolf Oppenheimer. Strahlentherapie, 1928, XXIX, 315.

In the author's opinion, moderate doses combined with massage bring relief in a considerable number of cases of hypertrophic prostatitis. Such treatment can not prevent the progress of the disease, particularly in the development of chronic retention. In some cases of bleeding, roentgen therapy was of help. High doses may aggravate an existing cystitis. It may make a future operation difficult and delay healing of the wound. *Technic:* 3 mm. Al., 24 cm. F.S.D., 33 to 40 per cent E.D., through a perineal field, on two successive days; repetition at intervals of two to four weeks.

E. A. Pohle, M.D., Ph.D.

Experience with Glucose Therapy Combined with Roentgen Rays in Malignant Disease (after E. G. Mayer). H. Füllsack. Strahlentherapie, 1928, XXVIII, 795.

A few years ago, Mayer made a suggestion to treat inoperable tumors by roentgen rays, at the same time injecting hypertonic glucose solution intravenously. The author tried this procedure in 35 cases and could not see any beneficial effect of this combined therapy.

E. A. Pohle, M.D.

Pyelography: Errors in Technic and Interpretation. Miley B. Wesson. Calif. and Western Med., November, 1928, XXIX, 297.

Because pyelography and even simple cystoscopy is still held responsible for an occasional death and hence must not be regarded as a harmless maneuver, in this paper an attempt is made to emphasize the simplicity and exactness of the various steps, and at the same time to call attention to the dangers that beset the path of the bold but untrained novice.

Dr. Wesson advises the use of 12 per cent

sodium iodid, and the use of double pyelograms except when specifically contra-indicated. Bilateral pyelograms are very important as they permit the comparison of the two sides and constitute an economic factor for the patient. He states that a double pyelogram is merely a pelvic lavage with harmless 12 per cent sodium iodid, a film being made during the process, and is really less dangerous than a lavage with 1 per cent silver nitrate or 3 per cent mercurochrome.

Double pyelograms can be taken with impunity in any case that has been subjected to bilateral catheterization.

Pyelography is usually a simple "fool-proof" procedure, but cystoscopy is not, and complete anuria and death follow urethral instrumentation and are then attributed to the lavaging of the kidney pelvis with sodium iodid —a harmless intravenous agent.

F. B. Sheldon, M.D.

An Unusual Type of Supernumerary Digit. S. H. Corrigan. Can. Med. Assn. Jour., September, 1928, XIX, 342.

The author can find no similar case on record. The digit is attached by a small pedicle to the external lateral aspect of the proximal phalanx of the little finger. A photograph is shown.

L. J. Carter, M.D.

The Esophylaxis of the Skin from the Viewpoint of the New Light Research. Alois Memmesheimer. Strahlentherapie, 1928, XXIX, 1.

In 1916, E. Hoffman suggested that radiation may stimulate the inner secretion of the epithelium of the epidermis. It would be possible to further the healing of tuberculosis by forming or increasing antibodies. In memory of his sixtieth birthday, the author presents a brief discussion of this theory and points out that quite a number of phenomena can be explained by esophylaxis.

E. A. Pohle, M.D.

Follicular Dental Cyst: Operation. Henry M. Goodyear. The Laryngoscope, May, 1928, XXXVIII, 328.

The author reports a case of follicular dental cyst. The patient was colored, aged 21, and came to the hospital with a temperature of 104 degrees. Wassermann reaction was negative. There had been a swelling over the left antrum for twelve years, gradually enlarging, without pain, except during acute coryza. Roentgenogram revealed one tooth in the lacrimal angle and one well up in the lateral anterior wall, with the crowns pointing toward the center of the tumor mass. The patient was operated on five days after entrance and the entire tumor mass (about 4 inches in diameter) removed. Healing was prompt. This tumor is an unusual type, involving the maxillary sinus.

B. C. Cushway, M.D.

Further Observations on the Saturation Method in Roentgen Therapy as Applied to Deep-seated Malignant Disease. George E. Pfahler. Am. Jour. Roentgenol. and Rad. Ther., September, 1928, XX, 233.

The "saturation method" in roentgen therapy consists in administering to the area to be treated an erythema dose which is to be followed for a short period of time by smaller doses at frequent intervals to maintain the effect and just offset the rate of loss. Pfahler's technic consists in administering 80 to 100 per cent of an erythema dose through each portal on successive days and in maintaining by small additional doses, administered daily, the saturation point for a period of from 10 to 14 days. This plan of therapy is based on the assumption that the chief desirable effect of the radiation depends upon its direct destructive effect on the tumor cells and that by maintaining the saturation effect throughout the period of cell division, the tumor will be more completely destroyed.

Advantages of the saturation method are: Delivery of maximum radiation in the shortest period of time, with resultant maximum de-

structive effect; irradiation of all cancer cells during cell division; avoidance of treatment reactions by dividing the dose, and, in the experience of the author, clinical results superior to those obtainable by any other radiation method.

J. E. Habbe, M.D.

Regarding Light Therapy of Erythrocyanosis Crurum Puellaris. Alfred Perutz. Strahlentherapie, 1928, XXIX, 283.

Erythrocyanosis crurum puellaris, which is, perhaps, identical with the chronic erythema nodosum (Deneke), has attracted the attention of dermatologists lately. The author has observed eight cases of this type—three cases were treated by X-ray, four by ultra-violet rays, and one by infra-red rays. A dose of from one-half to one-third E.D., filtered through 0.5 Al., was applied, and repeated after four weeks. Two of the cases treated by X-rays responded well, one not at all. Following the exposure to ultra-violet rays, the itching sensation disappeared temporarily. The same result was observed after treatment by infra-red radiation.

E. A. Pohle, M.D., Ph.D.

The Treatment of Carcinoma of the Breast. Karl Buchholz. Strahlentherapie, 1928, XXIX, 698.

The author presents a statistical discussion of the cases of breast carcinoma going through the surgical clinic from 1906 to 1924, covering a total of 384 patients. One surgeon operated on all cases, while the technic of radiation changed with the development of the X-ray apparatus. Very often radium was given in addition to roentgen rays. Of the total of 384 patients, 358 were operated on, 3 of the remaining patients refused operation, while 23 were inoperable. These 26 patients are not considered in the statistics. One hundred twenty-one cases were operated on only; 237 received X-ray or radium or both following operation. The numerical facts of the entire material after three to ten years show that operation plus radiation leads to 100 per cent better results than operation alone. The postoperative treatment of carcinoma of the breast with moderate doses is, therefore, recommended.

E. A. Pohle, M.D., Ph.D.

On the Accuracy of Calibrating Standard Instruments with the Three-electrometer Method. H. Hase and H. Küstner. Strahlentherapie, 1928, XXIX, 730.

The sources of error which may occur in the two-electrometer method are pointed out, suggestions for their remedy being given. The authors also present a three-electrometer method which permits an accurate calibration of standard instruments in *R*-units. It is shown, for instance, that a percentage error of 50 per cent in the two-electrometer method will lead to an error of only 1.6 per cent with the three-electrometer method.

E. A. Pohle, M.D., Ph.D.

Potential, Filter, Half Value Layer, Homogeneity, and Depth Doses in Deep Therapy. Hans Jacobi and Adolf Liechti. Strahlentherapie, 1928, XXIX, 503.

The authors have continued their experiments regarding the problems in dosimetry and present the following conclusions:

1. The sphere gap is a suitable means of measuring potential in diagnostic work and therapy if proper precautions are used. The relation between half value layer and potential and filter is illustrated in curves.

2. The half value layer is practically independent from type of tube and form of current. It is controlled only by potential and filter. This assumes, however, that the average thickness of the glass walls of the tubes is equivalent to about 3 mm. aluminum filtration.

3. The safest way to define quality is the direct determination of the half value layer. In practice, it is possible, however, to get a definite idea of the distribution of X-ray intensity in the body based on an accurate measuring of potential and filter.

4. The depth doses are dependent upon the

half value layer and to some degree upon the homogeneity; they are entirely independent from the current curve.

E. A. POHLE, M.D., PH.D.

Large Pulmonary Abscesses Caused by Pyogenic Micro-organisms. F. Bezancon and A. Jacquelin. Paris Medical, Feb. 16, 1929, p. 167. (Reprinted by permission from the British Medical Journal, April 6, 1929, No. 3561, p. 59 of Epitome of Current Medical Literature.)

During the past few years the subject of large pulmonary abscesses has received much attention, especially as a result of the War and of the epidemics of influenza. The authors claim that they should be regarded as a clinical entity, and that X-rays and bronchoscopy have shown that this type of abscess is neither so rare nor so fatal as had previously been thought. They recognize three main types: (1) the acute circumscribed, by far the most common; (2) the extensive and chronic, with successive attacks and frequently fatal, and (3) the extensive acute abscess with a tendency to diffuse necrosis, associated with high bacterial virulence, pneumonic in type, and very fatal. The first recognizable sign of such an abscess is the expectoration of pus, examination of which is of prognostic value. The physical signs vary, and may indicate a cavity in the lung; sometimes a layer of fluid due to pleural effusion masks the deeper trouble. Often there is only dullness, and sometimes, in the deep central forms, signs are absent. X-ray examination usually shows one large abscess cavity, often partly filled with air; sometimes the cavities are double or multiple. This type of abscess generally clears up in from two to three months. The second or chronic type is distinguished by the failure of medical treatment, a history of repeated attacks of bronchitis, and the insidious onset. The sputum is more frothy and mucous, dyspnea is more marked, and cough is a more important feature. Radiography shows several abscess cavities, often in cluster formation. Pleural adhesions and contracting fibrous bands are the rule, so that pneumothorax is likely to be ineffective, and thoracotomy must be considered preferable. The general signs of this type are successive attacks of pyrexia, coughing, and increased expectoration, often accompanied by hemoptysis. The patient becomes emaciated slowly, and this variety may be termed pseudo-tuberculous. The third type is rare, and is usually caused by the pneumobacillus and the streptococcus. Clinically and anatomically it is a massive necrosis of lung tissue, beginning with a pneumonic invasion and followed by necrotic disintegration. The duration of the illness is from five to eight days. As regards the prognosis, pneumococcal abscesses tend to end in spontaneous cure; streptococcal abscesses sometimes clear up with remarkable quickness, but at other times lead on to chronicity or necrosis; while Friedlaender's pneumobacillus causes the most serious conditions.

The authors state that, owing to the chance of spontaneous cure in many cases, only medical remedies should be tried at first, such as eucalyptus or intravenous injections of urotropine. Serotherapy or autogenous vaccinotherapy are sometimes useful, and the thirst cure has given good results. Postural drainage has not been clearly successful in the authors' cases. The indications for surgical methods of treatment are discussed; first, pneumothorax, which, though often unsatisfactory, should be attempted. In certain cases phrenicectomy is useful, and in the chronic form, owing to the gravity and incurable nature of the disease, pneumotomy and thoracoplasty may be advisable.

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